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PHYSIOLOGICAL PRE-DETERMINATION[1]: THE INFLUENCE OF THE PHYSIOLOGICAL CONDITION OF THE SEED UPON THE COURSE OF SUBSEQUENT GROWTH AND UPON THE YIELD.

I. THE EFFECTS OF SOAKING SEEDS IN WATER.

BY FRANKLIN KIDD, M.A. (CANTAB.), D.SC. (LOND.)
AND
CYRIL WEST, D.SC. (LOND.), F.L.S.

(With Plates I and II.)

INTRODUCTION.

IN a survey of the literature of the last fifty years dealing with the Seed it is interesting to note that attention has been almost entirely concentrated upon the question of germination. The effect upon germination of every manner of treatment and of every sort of condition has been investigated in the case of the seeds of a great range of cultivated and wild plants.

In only a very few cases has attention been directed upon the question suggested by the title of this paper, namely, as to how far the physiological condition of the plant in the seed-stage (*i.e.* in maturation, dormancy and germination) may exert a pre-determining influence upon the whole subsequent course of growth and development. It is obvious from the point of view of crop production that this question as to the quality and size of the plants produced from any sample of seed may be even more important than the question as to what percentage of the sample is capable of germination. The degree of importance to be attached to this question appears when it is said that the yield from an equal number of plants of a pure line may vary to an extent of 50 per

[1] Balls, L. W. 'The Pre-Determination of Fluctuating Characteristics,' *Proc. Cambridge Phil. Soc.*, 1914; also, 'Analyses of Agricultural Yield,' Part III; 'The Influence of Natural Environmental Factors upon the Yield of Egyptian Cotton,' *Phil. Trans. Roy. Soc.* (Lond.), B, 208, 1917.

cent., or even more, owing to the pre-determining influence upon the resultant plant of the physiological condition of the seed as effected by environmental conditions both previous to and also during germination.

The plant physiological problem which we are raising and with which we propose to deal in this and in following papers under the same general title, may be stated formally at the outset as follows: Presuming always a given equal number of plants, what are the limits of variation in growth and yield which may be pre-determined by the action of environmental conditions during the seed stage both previous to and during the course of germination; and how far may later environmental conditions during the course of growth affect the latitude of such variation?

The experimental work conducted by the authors dealing with the problem thus outlined is in progress. The present paper deals with the first results of experiments designed to test the effect of preliminary soaking of the seed upon its subsequent growth and development[1].

In the next paper of this series the literature bearing directly or indirectly upon the subject will be reviewed. This review will clearly indicate the wideness of the field and the possibility of obtaining important results if experimental work on a sufficiently large scale be carried out both in the field and in the laboratory.

The stage of germination is obviously a very critical one in the life-history of the plant. By artificial control of the environmental conditions of the seed previous to and during the process of germination the cultivator may aim not only at the natural optimum, that is the yield produced by good seed under the most favourable conditions, but may even improve upon this.

EXPERIMENTAL.

Experiment 1. *Seeds sown on damp sand.*

Seeds of the following plants were used in this experiment, viz. culinary pea (*Pisum sativum*), dwarf bean (*Phaseolus vulgaris*), barley (*Hordeum*), and sunflower (*Helianthus annuus*). The seeds were soaked under 4 cm. of distilled water at an average temperature of 17° C. (*i.e.* the temperature of the laboratory) for periods varying from 8 to 72 hours and the result of this treatment upon the germination (Table I) and upon the subsequent growth of the same seeds (Table II) was observed. In this experiment both germination and growth occurred with the seed lying on damp sand in porous pots surrounded with water and loosely covered with glass.

[1] The experiments described in this paper were carried out during the autumn of 1917.

TABLE I. *Germination results.*

Name of plant	Number of seeds used	Germinations: 2 days after sowing. Previously soaked: 0 hrs	8 hrs	24 hrs	48 hrs	72 hrs	3 days after sowing. Previously soaked: 0 hrs	8 hrs	24 hrs	48 hrs	72 hrs
Pisum	25	0	15	23	17	11	7	22	24	22	16
Phaseolus	25	0	18	18	8	5	10	24	23	15	5
Hordeum	50	31	48	43	41	26	46	49	48	49	37
Helianthus	25	11	14	10	10	6	22	20	15	21	10

The above table shows that soaking the seeds for periods exceeding 24 hours in general diminishes the rate of germination.

TABLE II. *Growth results.*

Name of plant	Hours soaked	No. of seeds	After 12 days on damp sand at 17° C.: No. of plants	Average length of stem	Average length of root
Pisum	0	25	25	7·2 cm.	11·0 cm.
	8	25	23	7·0	8·2
	24	25	24	7·5	5·9
	48	25	18	3·0	2·0
	72	25	17	2·2	1·6
Phascolus	0	25	23	6·4	7·4
	8	25	19	3·5	3·7
	24	25	15	1·3	1·1
	48	25	1 (very feeble)	—	—
	72	25	0	—	—
				Dry weight (tops only)	
Hordeum	0	50	50	0·555 g.	
	8	50	50	0·510	
	24	50	44	0·455	
	48	50	50	0·435	
	72	50	41	0·395	

Pisum. The soaking of seeds of *Pisum sativum* previous to sowing has a marked effect upon the subsequent course and vigour of development. The number of plants produced is not affected by soaking the seeds for periods of less than 24 hours. The retarding effect, however, upon the subsequent growth shows itself in the case of the roots of plants produced from seeds soaked even for the short period of 8 hours. After 48 hours soaking of the seed it is very marked. The size of the shoots is not greatly affected unless the period of soaking has exceeded 24 hours.

Phaseolus. In the case of *Phaseolus,* soaking the seeds previous to germination has an even more marked effect upon the subsequent course and vigour of development. Soaking for only 8 hours reduces the total growth after 12 days to 7·2 cm. as compared with 13·8 cm. for those sown dry on damp sand in spite of the fact that those which were soaked had a considerable start in germination and sprouted earlier.

The second point of importance brought out by these experiments is that the striking effects of soaking the seed upon its subsequent development are not visible in first observations upon germination. For instance, the seeds soaked for 24 hours showed a full percentage of germination and at first the radicles of these seeds appeared to be the largest and strongest of the whole series. At this stage the seedlings were such as would obviously be selected for physiological experiments, for example, on the rate of growth as influenced by temperature, etc. Nevertheless, on the twelfth day of the experiment half these plants had died and the remainder showed only about one-sixth of the growth of those plants which had arisen from seeds not soaked previous to sowing.

Hordeum. There is a progressive decrease in dry weight with time of soaking of seeds previous to sowing. The seeds, which had not been soaked at all, gave rise to the heaviest plants after 12 days in spite of the fact that during the first 4 or 5 days they were obviously behind the others owing to their slower germination.

Experiment 2. *Seeds sown in soil.*

In this experiment the following seeds were used:

Wheat (Sutton's, Sept. 1917),
Oats (Sutton's, Sept. 1917),
White Mustard [*Brassica alba*] (Carter's, Sept. 1917),
Dwarf Bean [*Phaseolus vulgaris*] (Sutton's, Sept. 1917),
Broad Bean [*Vicia Faba*] (Carter's, Nov. 1916),
White Lupin (Carter's, March, 1916).

Samples of the above-mentioned seeds were soaked for the following periods, 6, 24, 48 and 72 hours respectively under conditions similar to those of the previous experiment. They were then sown in potting soil in a greenhouse with an average temperature of 65° F., and were transferred to a greenhouse with an average temperature of 55° F. as follows: White Mustard after 4 days, Wheat and Oats after 5 days, Lupins after 7 days, Dwarf Beans and Broad Beans after 9 days.

The results obtained after 7 days are given in the following table:

TABLE III. *Germination results.*

Name of plant	Number of seeds used		Seed soaked previous to sowing				
			0 hours	6 hours	24 hours	48 hours	72 hours
Wheat	100	Germination results	Average height of seedlings 3 cm.	Average height of seedlings 3 cm.	Average height of seedlings 5 cm.	Average height of seedlings 5 cm.	Average height of seedlings 5 cm.
Oats	100		Few just sprouting	Average height 2 cm.	Average height 2 cm.	Average height 5 cm.	Average height 1 cm.
White Mustard	100		Average height 4 cm.	Average height 6 cm.	Average height 5 cm.	Average height 5·5 cm.	Average height 5 cm.
Dwarf Bean	25		0 showing above ground	4 showing above ground	1 showing above ground	0 showing above ground	0 showing above ground
Broad Bean	10		0 showing above ground	0 showing above ground	5 showing above ground	4 showing above ground	3 showing above ground
White Lupin	25		3 showing above ground	18 showing above ground	25 showing above ground	25 showing above ground	19 showing above ground

The results showing the effect of the various periods of soaking are given under separate headings for each crop.

Wheat. The five cultures were photographed after 4 weeks' growth (Pl. I, fig. 1) and showed a progressive increase in height from No. 0 (seed not soaked previous to sowing) to No. 2 (seed soaked 48 hours previous to sowing), but in No. 3 (seed soaked 72 hours) the plants were smaller than those in No. 2 and were obviously less vigorous. When the dry weight of the tops was taken (Table IV) the results confirmed this observation. The dry weight of the plants from seeds soaked for 48 hours was approximately 10 per cent. greater than that of the plants from seeds sown dry.

It appears also that the period of soaking bears no relation to the percentage of germination.

TABLE IV. *Growth results.*

WHEAT

No. on Plate-fig.	Hours soaked	No. of germinations	Dry weight per 100 seedlings (tops only) after 5 weeks' growth
0	0	94	2·148 g.
¼	6	92	2·065
1	24	96	2·119
2	48	95	**2·369**
3	72	97	2·206

Oats. The oats were harvested 3 weeks after sowing. They show a result similar to that of the wheat. The number of germinations is not influenced by the period of soaking, but the dry weight of the seedlings from seeds soaked for 48 hours is 62 per cent. greater than that of the seedlings produced from seeds sown dry.

TABLE V. *Growth results.*

OATS

Hours soaked	No. of germinations	Dry weight per 100 seedlings (tops only) after 3 weeks' growth
0	97	0·665 g.
6	96	0·966
24	95	0·954
48	99	**1·075**
72	96	0·833

White Mustard. The white mustard was harvested after a fortnight's growth. In this case the plants giving the highest dry weight were those from seed soaked six hours. The others form a graded series. The plants from the seeds soaked for 6 hours are about 25 per cent. heavier, but those from seed soaked for 72 hours are about 8 per cent. lighter than those produced from the dry seed. The number of germinations is apparently not affected by the soaking treatment.

TABLE VI. *Growth results.*

WHITE MUSTARD

Hours soaked	No. of germinations	Dry weight per 100 seedlings (tops only) after 14 days' growth
0	96	0·535 g.
6	88	**0·657**
24	93	0·577
48	77	0·571
72	100	0·479

Dwarf Beans (*Phaseolus*). These plants were harvested $4\frac{1}{2}$ weeks after sowing. Figs. 2 and 3, Plate I, show the appearance of the series at the time of harvesting. As in the previous experiment, even the shortest period of soaking (*i.e.* 6 hours) has a marked pre-determining influence in retarding growth in the course of later development to the extent of 26 per cent. as compared with the plants produced from seed sown dry, although both the number of germinations and the number of plants resulting are not affected. Preliminary soaking of the seed for 1 day

exercises a retarding influence upon the growth of the plants produced after a month's development to the extent of nearly 60 per cent. Longer periods of soaking has an even greater effect, whilst soaking for three days kills all the seed.

TABLE VII. *Growth results.*

DWARF BEAN

Hours soaked	No. of germinations resulting in plants	Dry weight per 100 seedlings (tops only) after $4\frac{1}{2}$ weeks' growth
0	22	27·41 g.
6	23	20·41
24	6	11·42
48	7	9·63

Broad Beans (*Vicia Faba*). Observations on the effect of the soaking treatment of the seed upon the subsequent development of the plant were made three weeks after sowing. The small number of these larger seeds used in the experiment does not allow us to place so much value on the results obtained. Those sown dry and those sown after only six hours' soaking did not complete their germination and moreover, the plants produced from the seeds which did completely germinate were very poor compared with those from seeds soaked 24, 48 and 72 hours respectively. The interest lies in the fact that a diametrically opposite result of soaking the seed appears in the subsequent growth of this variety of bean as compared with that obtained with the Dwarf Bean (cf. Table VIII with Table VII).

TABLE VIII. *Growth results.*

BROAD BEAN

Hours soaked	No. of plants produced after 3 weeks in soil	Average height of plants after 3 weeks' growth in soil
0	7	4 cm.
6	4	3·5
24	10	8·4
48	10	8·4
72	10	10·4

White Lupin. In every member of the series the plants showed very uneven growth, but the effect, if any, of the soaking treatment of the seed upon the plants produced was not apparent to the eye.

Experiment 3. *Effect of Temperature during soaking period.*

In this experiment the influence of the temperature obtaining during the period of soaking upon the degree and nature of the pre-determining influence of soaking upon subsequent growth and development was investigated.

25 seeds of Dwarf Bean (*Phaseolus vulgaris*) (Sutton's, Sept. 1917) were used in each experiment.

The seeds were soaked in distilled water for periods of 6, 24, 48 and 72 hours respectively at four different temperatures, viz.:

1. 10° C. (average)
2. 20°
3. 25°
4. 30°

} Thermostat } In the dark.

After the soaking treatment the seeds were sown in good potting soil in large wooden boxes which were placed in a greenhouse with an average temperature of 55° F.

The following table gives the results obtained when the beans were harvested 3 weeks after sowing:

TABLE IX. *Germination and growth results.*

DWARF BEANS

Hours soaked	Dry weights per 100 plants produced from seeds soaked at 10° C.	20° C.	25° C.	30° C.
0	(24) 23·27 g.	(24) 23·27 g.	(24) 23·27 g.	(24) 23·27 g.
6	(10) 21·34	(16) 21·47	(17) **22·17**	(20) 21·74
24	(9) 20·05	(12) **21·04**	(18) 20·33	(16) 19·18
48	(8) 13·92	(15) **17·93**	(20) 16·28	(13) 11·54
72	(15) **17·80**	(16) 16·28	(12) 12·26	(0) —

The figures in brackets denote the number of plants produced.

The most obvious result of this experiment is that a deleterious effect is visible even after the shortest period of soaking (*i.e.* 6 hours) and that this deleterious effect progressively increases with longer periods of soaking at all temperatures.

The second result of importance from an economic point of view is that the temperature during the soaking treatment especially for the shorter periods does not have any *marked* effect upon the figures for the dry weight per plant.

Upon the number of germinations, however, and consequently, upon

the net yield, the effect of temperature during the period of soaking is most striking as is shown by the following table:

TABLE X. *Germination and growth results.*

Period of soaking	Temperature during the soaking treatment	Total No. of germinations	Actual dry weights of the plants produced	Dry weights per 100 plants
1 day	10° C.	9	1·805 g.	20·05 g.
1 day	20° C.	12	2·525	21·04
1 day	25° C.	18	3·660	20·33
1 day	30° C.	16	3·070	19·18

Considering these results from a plant-physiological point of view, it is curious to note that we do not obtain the simple result which might have been expected, namely, that both number of germinations and yield per plant should fall off, with increasing time of soaking, most gradually at low temperatures and most rapidly at high temperatures; in other words, we might have expected the highest values for plant weight and number of germinations at the lowest temperature for any given time of soaking. In Table IX the highest figures obtained for any given time of soaking are printed in heavy type and show at a glance that this is not the case.

Experiment 4. *Seeds sown on damp sand.*

In Figs. 4 and 5 (Plate II) the results of further experiments with another variety of Dwarf Bean are shown. In these cases a much longer subsequent growth period (*i.e.* 8–10 weeks) was allowed. The difference in the results from seeds sown dry (Figs. 4 A and 5 A) and seeds which had been soaked for 12 hours previous to sowing (Figs. 4 B and 5 B) is very marked.

Removal of the testas before soaking apparently has no effect upon the results of the soaking treatment (Figs. 5 A and 5 B).

DISCUSSION AND SUMMARY.

Three main conclusions may be drawn from the results obtained from the experiments described in this communication, although these were not carried through to the final stages of development of the various plants concerned. In the first place, soaking the seed in distilled water previous to sowing may have a marked effect upon the subsequent growth of the plant. In the second place, a germination test cannot be relied upon in the least to give any criterion of what this effect may be. And thirdly, the nature of the effect is strongly specific, quite different

results being obtained by similar treatment upon closely allied plants. Beans, which give the most striking results, illustrate these three points effectively.

Firstly, the result of soaking Dwarf Beans (*Phaseolus vulgaris*) even for a period of 6 hours becomes evident after a month's growth by a decrease in the dry weight yield of the tops of the plants of 26 per cent. as compared with the control crop produced from seeds planted dry (Exp. 2). Secondly, seeds of the same species, which were soaked for a period of 24 hours before sowing, showed by far the most rapid and vigorous germination (Exp. 1), while later, 12 days after sowing, the total length of these plants as compared with the controls set dry was as 2·4 : 13·8. Lastly, when the results obtained with the Broad Bean (*Vicia Faba*) are compared with the above (Exp. 2), an absolute reversal in the effects following soaking is observed. In the case of the Broad Bean the effect of soaking the seeds for periods up to 3 days was found to be increasingly beneficial, not only on the percentage of germination, but also upon the size of the plant produced. Thus, after an interval of 3 weeks the average height of the plants produced from seeds planted dry was only 4 cm. as compared with 10·4 cm. in the case of those soaked for 3 days before sowing.

The thanks of the authors are due to Mr W. Hales, A.L.S., Curator of the Chelsea Physic Gardens, for assistance in the culture of the seedlings.

EXPLANATION OF PLATE FIGURES.

PLATE I

Fig. 1. Wheat plants from seeds sown dry (0), soaked in water previous to sowing for periods of 6 hours ($\frac{1}{4}$), 24 hours (1), 48 hours (2), and 72 hours (3) respectively. Photographed 4 weeks after sowing.

Fig. 2. Dwarf bean plants from seeds sown dry (0), soaked in water previous to sowing for periods of 6 hours ($\frac{1}{4}$), 24 hours (1), and 48 hours (2) respectively. Photographed $4\frac{1}{2}$ weeks after sowing.

Fig. 3. A parallel series to that shown in Fig. 2. Photographed $4\frac{1}{2}$ weeks after sowing.

PLATE II

Fig. 4. A. Dwarf bean plants from seeds sown dry.
B. Dwarf bean plants from seeds soaked for 12 hours previous to sowing. Photographed 8 weeks after sowing.

Fig. 5. A. Dwarf bean plants from seeds sown dry. The testas were removed.
B. Dwarf bean plants from seeds soaked for 12 hours previous to sowing. The testas were removed previous to the soaking treatment. Photographed 8 weeks after sowing.

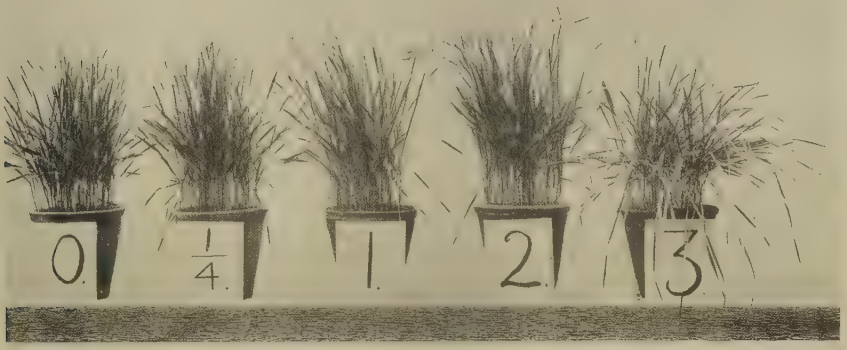


Fig. 1

Fig. 2

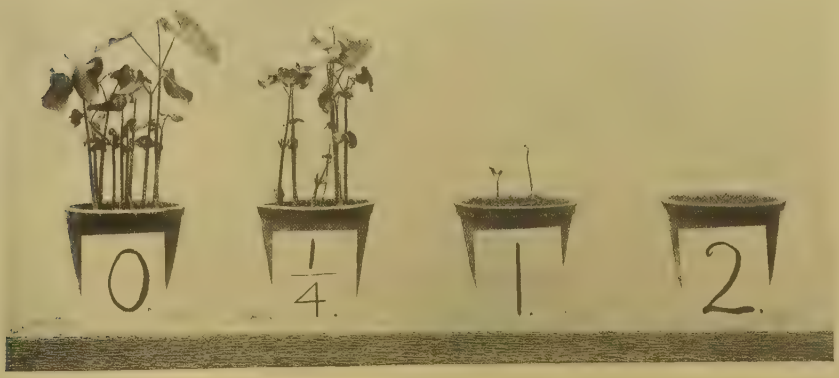


Fig. 3

A Fig. 4 B

A Fig. 5 B

"REVERSION" AND RESISTANCE TO "BIG BUD" IN BLACK CURRANTS.

By A. H. LEES, M.A.

(*Plant Pathologist, Agricultural and Horticultural Research Station, Long Ashton, Bristol.*)

(With Plates III—VI.)

In this paper owing to the apparently close connection between Reversion and Big Bud it is proposed to treat the two subjects together. It seems impossible to explain Reversion without discussing Big Bud and special cases of the latter necessitate for their explanation and therefore for the explanation of certain cases of Reversion, reference to the factors governing resistance to Mite attack.

For these reasons it has been necessary to combine in one paper material that would otherwise have been better suited for two.

REVERSION.

This disease is known under various names, such as "Going wild," "Running off," "Nettle leaf" and possibly others. "Reversion" seems now to be the commonly accepted name and, being so, is used here. The name of course implies that the plant is reverting to the wild type. Scientifically the idea is extremely improbable and there is nothing to show from the observed facts that such a process has been or is going on. For lack of a better name however the term reversion will be used in this paper.

Characteristics of the Disease.

In a fully reverted bush the following points may be observed.

(1) The fruit "runs off"; that is, at picking time either no berries are found on the strig or only a very few generally undersized fruits instead of the many plump ones of the normal bush. These few berries may be at the base or at the tip of the strig.

(2) There is an extensive growth of the laterals resulting in a crowded, instead of an open, form of bush.

(3) The internodes are abnormally long and thin.

(4) The leaf is sharp pointed, abnormally narrow, with a more than usually serrate margin.

While in badly attacked bushes all these points may be observed at once it is much more common to find only some of them.

The disease is nearly always gradual in its appearance and frequently parts of a bush will show signs while the rest is quite healthy. Until the eye becomes trained it is easy to pass over bushes which are only beginning to revert.

While from the practical point of view the failure of the fruit is undoubtedly the most important point it is not in the author's opinion the most crucial for the elucidation of the disease. If the theoretical explanations afterwards to be considered are correct it is clear that the extensive outgrowth of lateral wood precedes the running off in point of time and may serve to explain the latter. In discussing the disease therefore and its association with Big Bud or otherwise the facts will be considered as affecting the outgrowth of laterals and not the failure of the fruit.

Character of the normal growth in the Black Currant.

For a correct interpretation of the pathological condition it is necessary to have a correct conception of the normal, and therefore no apology is needed for a description of what is perhaps already familiar to most.

A strong healthy twig of the current year's growth shows the following arrangement of buds in winter.

The lowest buds are generally weak and are often wood buds, that is buds having one single growing point surrounded by rudimentary leaves. The rest of the buds up to the terminal are flower buds. They vary in strength but all show the same structure. They consist of a median flower rudiment, made from the typical growing point present early in the summer, and two accessory, weak growing points.

The terminal bud is a typical but strong wood bud.

In the subsequent year growth takes place as follows (Pl. III, Fig. 1). A strong wood growth issues from the terminal bud forming the fruiting wood for the following year and the flower rudiments in the lateral buds unfold and bear fruit. One or both accessory growing points present in the fruit bud make a short spur-like growth and form fruit buds to give fruit the following season.

The wood buds at the base of the winter shoot remain dormant, make

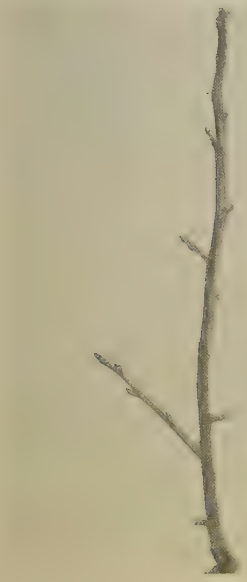


Fig. 1. Whole of last year's growth and base of current year's in a normal shoot from Long Ashton. Most of the flower buds of last year have made a spur-like growth from the accessory growing points. Note absence of development of top buds of the last year's growth due to their original weakness as shown in Graphs I and II.

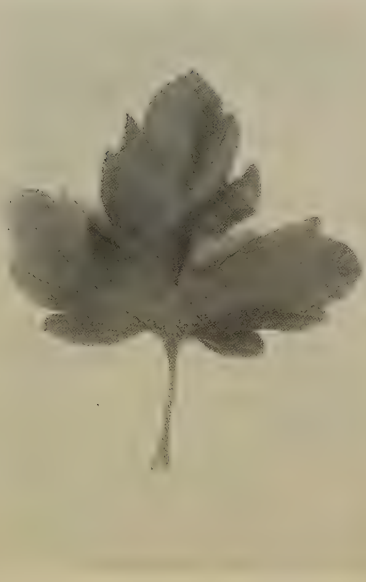


Fig. 2. "Oak Leaf" caused by mite attack on a strong growing terminal bud.

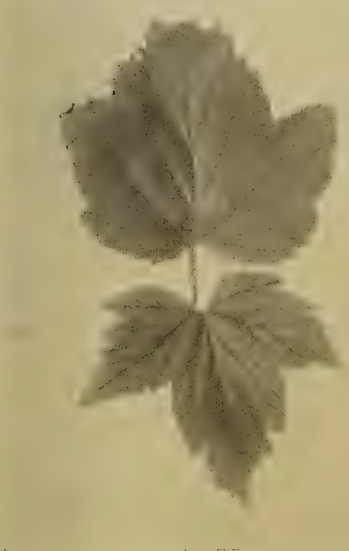


Fig. 3. A normal leaf, broad and blunt, and a "reverted" leaf, narrow and pointed.

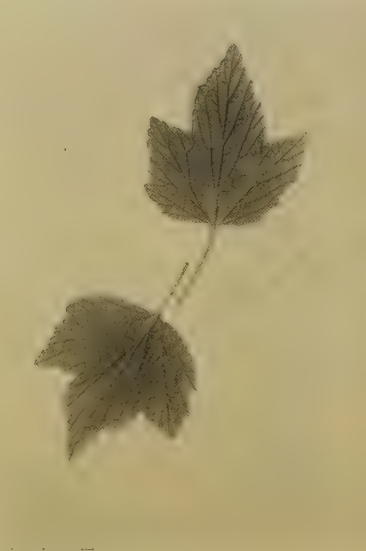


Fig. 4. Normal leaves formed at the end of the season. They are smaller and more divided than the leaf formed during active growth.

a short or a strong growth depending on the vigour of the bush as a whole. Usually they remain dormant or make a short growth.

The normal growth of the black currant may therefore be said to be terminal and correct pruning and culture ensures a constant supply of long straight young wood.

Character of abnormal, reverted growth.

Case 1. Associated with moderate or large numbers of Big Buds.

Since 1914 it had been noticed that Reversion often occurred amongst mite attacked bushes and frequent cases of such association came under the author's experience in visiting plantations in Worcestershire and Herefordshire. It was natural to suspect that the two were linked together in some way but the case was complicated by the fact that reversion often occurred amongst bushes where there was either no big buds or very few. A year or so after the connection had first been noticed, bushes began to revert at Long Ashton and it was possible to investigate the disease under good conditions. Here it was apparently associated with the Big Bud attack which had been increasing ever since the bushes were planted.

In order to discover how far the tendency to reversion corresponded with mite attack the bushes were carefully examined in June and marks given for the amount of reversion present. Four classes were recognised, normal bushes, slightly attacked bushes where the leaves tended to the pointed type, but with normal fruit, a worse condition where the fruit had partly run off and a still worse where the fruit had completely run off.

In the following winter the bushes were divided into four classes as regards numbers of big buds present. This was done for two years. In this time there were forty cases of reverted bushes in the two last classes which were also placed in the last two classes for Big Bud and only twelve such reverted cases that were placed in the second class for Big Bud (namely in the class showing a minimal number of big buds).

There was no case of definite reversion on bushes that showed no big bud the following winter. The evidence thus pointed to a close connection between the two diseases.

As was previously mentioned, one of the most characteristic signs of a reverted bush is the extensive growth of lateral wood, a condition which is usually associated with a lack of suckers from the base. This lateral growth proceeds largely from the lower buds of a yearly growth and not from the higher as occurs if the terminal be injured or removed

in summer time. Any cutting back of the terminal in summer time or hard cutting back in winter does not produce the same effect since wood growths then emanate from the buds immediately below the pruned part.

If the history of a mite attacked bush be followed in detail the following appears to happen in the case of a strong shoot. The terminal bud usually gets infested by migrating mites early in June. Generally only a few are present and the growth from the terminal appears normal. Should many mites succeed in getting in and the bush be in strong growing condition very characteristic leaves are developed. This type is generally known as "oak leaf" by growers. The leaf is thick and dark green and bears no resemblance to the usual shape of the normal leaf. It is a certain sign of a mite infested terminal (Fig. 2; cf. Figs. 3 and 4). In any case the growth from the terminal is checked to some degree.

This check is sometimes sufficient to force out some of the lateral buds lower down during the current year (Pl. IV, Fig. 5). The laterals of course will push according to their strength during the succeeding year. Most of them, where mite is fairly abundant, will also be mite infested, since they are formed at the time when mite migration is most active. They therefore cannot grow out at all. If any escape, and especially if they are not in the process of changing into fruit buds, as they would be normally, they will sometimes make a short outgrowth in the current year and in any case become stronger buds than they would otherwise be (Fig. 5). The lateral buds formed after June, that is those which during the next winter are situated just below the terminal, usually escape mite infection and develop into fruit buds having the structure already described.

The reason why these often fail to grow out under the stimulus of the check to the terminal bud may be explained thus. At the time of their formation the growth push of the plant is rapidly decreasing (see Tables I and II) and also that the two accessory growing points in the forming fruit bud are very weak. If however the check takes place comparatively early before the lateral buds have changed into flower buds out-growth often takes place (Figs. 5–8).

The position therefore at the end of the first year is that the terminal bud is "big" as also most of the lower laterals. A few of these have been abnormally strengthened or have made a slight outgrowth while the higher laterals are normal fruit buds.

The following year when growth begins the terminal, being mited, can make no further growth and there is consequently pressure put upon the buds below. As pointed out, the basal buds have been unduly

Fig. 5. Outgrowth of laterals formed during last year (about one inch of growth) due to strong mite check to terminal. In the current year growth has continued from these strengthened buds and shows a reverted type of foliage.

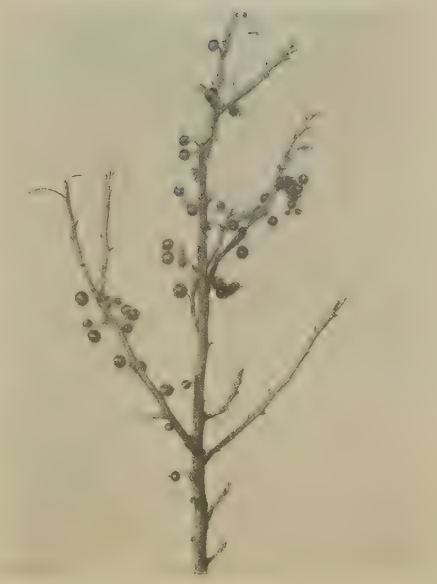


Fig. 6. Type of reverted growth at Long Ashton. Three years' growth on "French." Two year old wood bears fruit that has "run off." Forced lateral growth occurs each year either just below the then terminal or from wood buds low down on the year's growth.

Fig. 7. Shoot from Long Ashton photographed in September showing old terminal big bud (whitened to show up). In this case nearly all the buds below the terminal being mited only three shoots grew as shown in the figure The leaves are reverted in type.

Fig. 8. Shoot from Long Ashton photographed in December, showing result of check to terminal during *early* summer.

Table I. *Growth Graph of Seabrook's Black.*

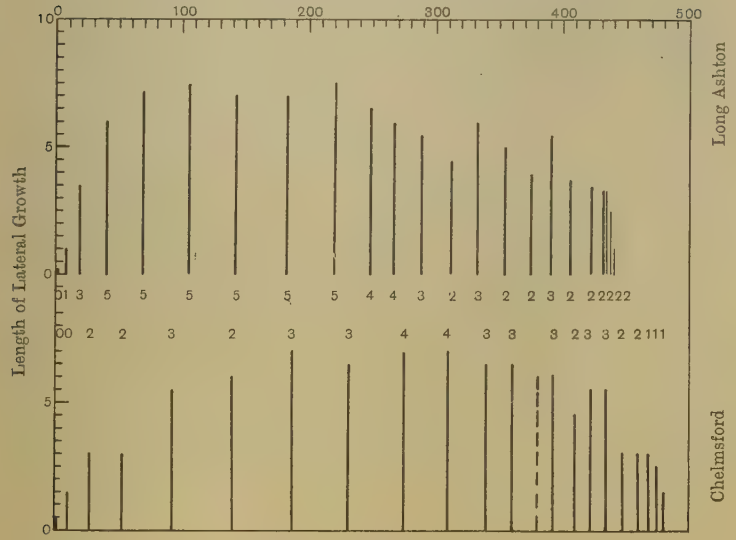
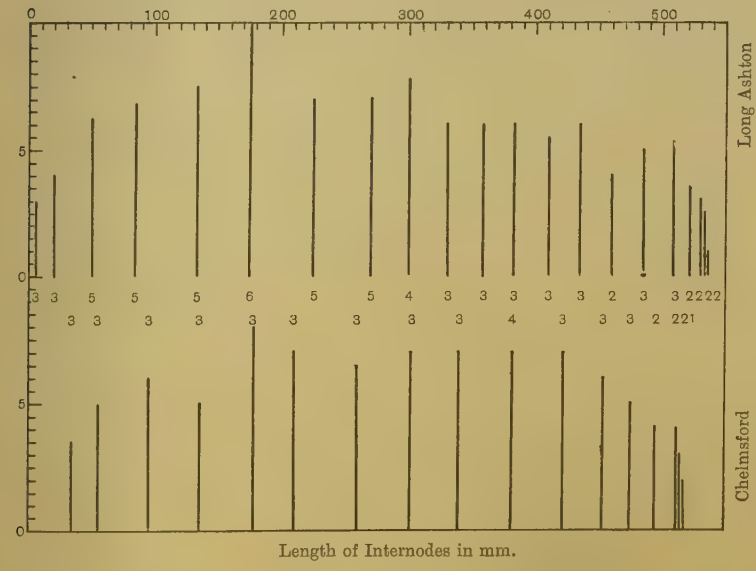


Table II. *Growth Graph of Boskoop Giant.*



strengthened during the previous season and it is they that now grow out into twigs of various lengths.

There is also often some growth made from one or both of the accessory growing points present in the highest fruit laterals. It is usually only slight and may be explained by the fact that though these growing points are weak the growth push of a fruit tree is always at its maximum at the top of the yearly growth. The previous year, when they failed to grow out, their growing points were still weaker and the growth push of the plant as a whole was decreasing, but in the spring of the current year not only are they stronger but the growth push of the plant is increasing to a maximum.

Weak shoots show a slightly different behaviour from that just described for a strong shoot. A weak shoot usually means that terminal growth ceases early in the season and that therefore when mites are fairly abundant nearly all the laterals become "big." Such shoots do not form wood buds at the base and therefore no growing out takes place from that region. Generally there is a small amount of outgrowth from any lateral bud that has escaped mite but often the shoot becomes entirely bare and all growth from it ceases.

Where mites are very abundant and the bush is still making strong growth a slight variation occurs from what has been described. Here the check to the terminal bud in the first year is so strong that it disappears altogether during the summer while growth is still active and the laterals immediately below are thus stimulated to strong outgrowth the same year. This case must be distinguished from the previous one where the terminal, though checked, continues growing during the current season and only fails to grow next season because it has become a big bud. As explained below in the section treating the growth graphs of the black currant removal of the terminal in the winter causes buds far below it to shoot while removal in the summer causes buds just below it to shoot.

Case 2. Reversion of a Big Bud resistant variety.

At first sight it would seem that if Big Bud were a necessary antecedent to Reversion, resistant varieties should be free from it. Through the kindness of Messrs Seabrooks of Chelmsford I have been enabled to study some very interesting cases of reversion on the variety known as "Seabrook's Black." As the term "resistant" implies the variety does not under ordinary circumstances become badly attacked. In the firm's experience some big buds are sometimes formed but they never increase seriously in numbers and the bushes as a whole remain practically free.

Reverted wood of this variety differed from Long Ashton material in

that the characteristic lateral outgrowths arose from fruit buds instead of from basal wood buds (Pl. V, Fig. 9). From comparative examination of material from Chelmsford and Long Ashton there appears to be a greater tendency to form wood buds at the latter place than at the former. This is probably connected with the different climates at the two places. The result is that outgrowths are more in number and weaker in growth since many weak buds are pushed instead of a few strong. In both cases however the effect of a bushy type of growth is produced instead of a thin type, though the manner of production is slightly different.

When such reverted shoots were examined it was found that many of the buds were abnormally small (Fig. 10). Instead of being the normal oval shape they were angular like an inverted V and were only 2 mm. in length instead of being about 6 mm. When such buds were dissected it was found that the growing point had been killed and only some brown dried up material left to mark its position.

From previous experience of growing points being killed by mite during the summer at Long Ashton it was impossible not to assume that the same cause had produced this effect, namely mite attack. This hypothesis was supported by the fact that big buds were often also present though these were confined to the lower portion of the shoot while the "killed" buds practically always occurred higher up. It is reasonable to look upon these "killed" buds as having been more strongly attacked or as being less resistant to mite than typical big buds and it is possible to trace a complete series of buds showing various stages of resistance to mite attack.

At the bottom of the series where presumably the mite attack is greatest and resistance is smallest come the "blind" buds (Fig. 10). In this case no trace of a bud can be seen though the scar of the leaf in the axil of which it should have been borne is clear. The bud above and below it are usually quite normal.

Next in the series come the inverted V-shaped buds. They are small and consist of only about half the normal number of scales surrounding the dead growing point. In this case presumably the attack was either later or less strong or the growing point was more vigorous. Such buds are not usually found low down on a shoot for reasons subsequently explained. The next stage is normal in shape but smaller than a healthy bud and shows a killed growing point. Such buds are not very common and indicate that the killing process took place later than the last stage but before any tendency to fruit bud formation had taken place.

The next stage consists of buds resembling "big buds" in shape but

much smaller (Fig. 10). The growing point has had time to change into a flower rudiment but the scale leaves are deformed, showing the characteristic semi-spherical shape of big buds. There is however no mite present though sometimes the flower rudiment shows brown areas where it has presumably been injured by mite. In extreme cases of this stage the flower rudiment has been killed and one of the accessory growing points has changed into a very weak flower rudiment.

The last stage is the "big bud." Here the attack is so mild or the growing point so strong that the latter is not killed but is stimulated to irregular growth. In this resistant variety they occur almost without exception low down on the yearly growth.

The terminal bud may be either a "blind" one or a "killed" one but is only in the most rare cases "big."

These killed buds cause an interesting pathological effect on the pith. Ever since the disease had first come under the author's notice certain curious brown marks in the pith have been observed. They occur as smaller or larger streaks in the pith of the main stems but appear to start as spots just below a lateral bud.

I am indebted to my colleague Mr O. Grove, Oenologist at Long Ashton, for his investigation of it. He failed to find any organism microscopically and also failed to obtain any living culture from such spots. It is improbable therefore that they are caused by any organism. Microscopically they appear as brown gummy masses and strongly suggest degenerative changes.

When examining this mited material from Messrs Seabrook it was found that such brown spots were always connected with killed or blank buds. In a few cases the very beginning of the process could be seen consisting of a small spot only, immediately under the killed growing point. Not in every case of a killed bud could the spots be seen but wherever there was a brown spot a killed bud could be found close by from which the browning had started. It would appear probable therefore that such brown spots are due to degenerative changes in the pith cells consequent on the sap flow to the killed growing point being stopped. The phenomenon is an effect of the disease rather than a cause.

The presence of these "blind" and "killed" buds on shoots of "Seabrook's Black" immediately suggests the reasons of its resistance. Strictly speaking it is not resistant but very susceptible, so much so that the mite kills the growing point in an attacked bud and as a consequence cuts off its own supplies and thus perishes. A few buds however become "big" and serve as a small source of infection the following year.

Fig. 9. Reverted wood from a Seabrook's Black grown at Chelmsford. Last year's terminal, situated in the photograph between two lateral growths, was killed by mite and its base whitened to show its position. This year's extension growth was formed from one of the high lateral buds.

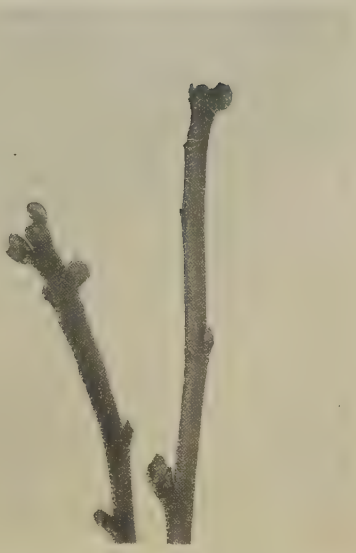


Fig. 10. Parts of two shoots of Seabrook's Black. In the longer shoot 1, 3, 4 and 5 (reckon from the top) are killed buds of varying size. Lateral no. 2 resembles a big bud but is smaller in shape and contains no mites. The terminal is blank, having been completely killed by mite. The smaller shoot is photographed partly end on to show the minute bud formed from one of the accessory growing points originally present in the now killed terminal bud. The shoot also shows "round" buds and "killed" buds lower down.

Fig. 11. Reverted wood of Seabrook's Black grown at Chelmsford. The immediate cause in this case was the formation of a flower bud as terminal in the preceding season. The strig of the fruit formed from it can be seen on the left-hand side at the extreme base of the current year's wood.

Fig. 12. Reverted wood of Seabrook's Black grown at Chelmsford. The strig formed from the flower bud formed as last year's terminal is clearly to be seen at the top of the figure. Nearly all the lateral buds have made abnormal growth.

At Long Ashton where the variety has been under observation for three or four years bushes have been found to be losing their resistance and one or two are showing a considerable number of big buds. There is no reason to doubt that the variety is under ordinary circumstances resistant and Messrs Seabrooks' experience supports this contention. Their experience however seems to be practically confined to the drier portions of England and it is possible that the variety might alter its behaviour in a wet climate. Thus it is noticeable that very few killed buds are present on Long Ashton examples and that big buds seem to have taken their place.

In order to study the method of growth of the variety at the two places two series of measurements were made. The length of each internode on typical shoots was measured and also the distance, for each lateral bud, from the apical end of the leaf scar of the subtending leaf to the highest scale scar on the lateral bud. This latter figure gives a fairly true idea of the "strength" of any particular lateral bud being, strictly speaking, the distance it has grown out since its inception. Tables I and II give the result of these measurements. The abscissae show the distance of the nodes in millimetres from the base of the current year's growth and also the length of the individual internodes. The ordinates show the relative length of growth made by the lateral buds and serve to indicate the strength of the individual buds. In order to make a clear graph these values are multiplied forty times. The figures running across the graph indicate the number of scale scars shown by the corresponding lateral bud. Table I represents the graphs of typical shoots of Seabrook's Black at Long Ashton and at Chelmsford. The shoots were chosen as nearly as possible equal in strength, the Long Ashton one having a diameter of 9 mm. at the base and the Chelmsford one $8\frac{1}{2}$ mm. The number of internodes also happens to be nearly equal, being 23 and 22. They may therefore be considered to be representative, especially as many other shoots from the two places showed the same general arrangement. The Chelmsford shoot showed a blank or blind bud at one node and this is indicated by the dotted line. It must be pointed out at the outset that the graphs are not necessarily strictly comparable in point of time; thus there is no indication that the two buds at about 185 were formed at the same time. Indeed it is known that growth continues at Long Ashton later than in drier climates, this period being in the case of apples three or four weeks. Looking first at the internodes in the Long Ashton specimen the normal summer check to growth begins at 220 and continues in a rather feeble way as indicated

by the comparatively long internodes until 430, the last three internodes being formed without any chance of elongation. It covers therefore a distance of eleven internodes. In the Chelmsford specimen the check begins at 330 and continues strongly, as indicated by the closeness of the internodes, until 460, a distance of eight internodes. The chief difference is therefore that the check at Long Ashton is gradual and weak while that at Chelmsford is sudden and strong. At the other end of the graph it may be noticed that the growth is more vigorous at Chelmsford than Long Ashton as the internodes are rather larger.

As a general rule the strength of the lateral buds is dependent on the vigour of the bush at the time of their formation and at first sight one would expect the Chelmsford figures to be higher than the Long Ashton ones whereas they tend to be slightly lower. An examination of the number of scale scars on the laterals, however, explains the situation. These numbers (running across the middle of the graph) show that whereas six at Long Ashton run as high as 5, at Chelmsford only two read 4 and it is clear from a superficial view that these average considerably less. From these figures one may draw the conclusion that the growth of laterals at Long Ashton continued over an extended period slowly, while that of the laterals at Chelmsford was characterised by a great push and a sudden check. The growth of the laterals therefore closely follows that of the internodes under the two different climatic conditions. These two kinds of growth can be very largely, if not entirely, accounted for by the different rainfall at the two places. Thus for a thirty-five year period the average monthly rainfall for June, July and August are shown in the accompanying table. July and especially August show a markedly increased rainfall at Long Ashton. The soil therefore in these months tends to be wetter and this tendency is increased owing to the factors encouraging evaporation, namely sunshine and air dryness, being less marked at Long Ashton than in the east of England.

TABLE III. *Rainfall in inches.*

	35 yr. av.	
Month	Chelmsford	Long Ashton
June	2·07	2·52
July	2·33	2·92
August	2·27	3·61
Total	6·67	9·05

Table II shows a similar graph for Boskoop Giant at the two places. In this case also the shoots are fairly comparable with each other and

with the Seabrook's Black graphs since they have a thickness at the base of 9½ mm. for the Chelmsford shoot and 9 mm. for the Long Ashton shoot. The influence of climate is here the same in all respects as in the Seabrook graph. The number of internodes however is less, being twenty-one and seventeen, though the length of the shoot is greater. The internodes are therefore, as can also be seen from a superficial glance, longer. Incidentally it may be remarked that Seabrook's Black is a better type for production of fruit, in that, in a given length of shoot, there are more lateral, that is fruit, buds than in Boskoop Giant. Comparing Seabrook's Black and Boskoop Giant under Chelmsford conditions the great concentration of internodes in the former at the end of the graph is in marked contrast to the corresponding part of the graph for the latter. It indicates an early check to growth, an indication which is borne out by the numbers of scale scars in the last nine laterals. These in the case of Seabrook's total eighteen while for Boskoop they are twenty-three. One may assume therefore that under the same climatic conditions Seabrook's receives a more decided check to growth than Boskoop.

If, as has been already assumed, the resistance of Seabrook's Black to Big Bud at Chelmsford is due to the fact that mites kill the bud growing points instead of forcing them into hypertrophied growth it should be possible to show that the conditions at Chelmsford are such as to bring this about more readily than at Long Ashton, where big buds are formed extensively on this variety. It has been shown above that the conditions at Chelmsford cause an early and marked summer check to terminal and lateral growth and therefore it is likely that mite attacked buds will have their sap supply very much curtailed at the time of this summer check. There will therefore be more tendency for these growing points to be killed by the mite attack than in the Long Ashton climate where growth continues longer. These facts also explain why some big buds are formed at Chelmsford and why they are practically always situated low down on the shoot. For it is just these buds that have been growing the longest and must have been, from their position, earliest attacked. A big bud therefore is formed from a growing point that has been attacked early and has had abundant and long continued sap supply while a killed bud is formed from a growing point that has been attacked late in the season and has a poor and brief supply of sap.

These considerations explain why some big buds are formed on Seabrook's and also why they do not increase in number. Probably it will be found that in wet years they do increase and in dry years disappear.

Transfer them however to a wet climate like Long Ashton and they continue to increase regularly and the variety apparently loses its resistance. If this theory is sound there should also be some explanation why Boskoop is not resistant at Chelmsford, since in that climate it has a more definite summer check than at Long Ashton. This is partly, at any rate, supplied by the fact that, as already pointed out, the summer check has much more effect on Seabrook's than on Boskoop. This habit of an early check to growth may be sufficient in itself to explain the mite-resistance but on the other hand there may be other factors which influence the result. There is however nothing in the evidence so far in the author's possession which would lead to any other conclusion. It would thus appear possible, by arranging the cultural conditions in such a manner as to get a marked and continued summer check, to cause "Big Bud" to disappear almost entirely from an infected plantation. Such a treatment will be tried in the near future.

The graphs of Table I serve to explain the character of the reverted wood in a typical shoot of Seabrook's Black. The terminal bud is either blind or a killed bud which means that it was attacked comparatively late in the season. The following year there is no strong terminal growing point to take the growth push so that this is distributed amongst the accessory growing points in the flower buds according to their strength and also according to their nearness to the apex of the shoot. The lowest buds (0 to 50) are not only weak but far from the apex and therefore are unmoved. The next two are stronger but being still fairly far from the apex usually make but a short growth. The next six are comparatively strong and usually make a considerable growth while the last five or six are so weak that, despite the extra push due to their nearness to the apex, they make but a short growth. Fig. 11 shows the kind of growth obtained in reverted shoots grown under Chelmsford conditions and agrees fairly well with the theoretical expectations.

Case 3. A. No Big Bud present. Killed buds extremely few or absent.

In addition to the material already described were shoots from Messrs Seabrook's from reverted bushes not showing any big buds. The terminals here were apparently normal and there were very few killed lateral buds. The fact that killed buds were to be found suggested the possibility that the original terminal had been killed and its place taken by one of the accessory growing points originally present in the terminal bud. This does sometimes undoubtedly happen, as may be seen in Fig. 10 which shows the killed terminal in a Seabrook's Black twig. The small bud immediately lateral to the blank terminal is one of the

accessory growing points. No such clear case could be found in the material coming under Case 3 and it is certainly not sufficient to account for the reversion there experienced.

Table IV.

Source of shoot	No. of terminals examined	Av. no. of internodes on shoot	Av. length of shoot in cm.	Av. length of internode in cm.
Sound Seabrook, Chelmsford				
Flower	17	15·5	32·0	2·06
Wood	12	16·6	49·9	3·00
Reverted Seabrook, Chelmsford				
Flower	29	15·8	38·3	2·42
Wood	4	16·7	41·7	2·50
Sound Seabrook, Long Ashton				
Flower	12	17·2	35·0	2·03
Wood	5	21·0	51·6	2·42
Sound Boskoop, Chelmsford				
Flower	—	—	—	—
Wood	10	17·9	54·1	3·02
Sound Boskoop, Long Ashton				
Flower	4	15·2	28·7	1·88
Wood	8	19·2	48·0	2·5

Examination of the terminals however suggested a possible cause. They were found in most cases to be flower buds. Thus (Table IV) out of thirty-three shoots examined twenty-nine had flower bud terminals and only four wood buds. In the spring these terminal flower buds would open and it would not be until an accessory growing point present in the bud was strengthened sufficiently that normal terminal wood growth could take place. While this process was going on however there would be a definite stimulus to growth on the buds situated below the terminal. In a fair proportion of cases the remains of the strig from the previous year's terminal flower bud (Figs. 11–13) could be seen and such an arrangement might easily lead to reverted wood being formed. In order to see how common this cause might be a number of shoots from different sources were examined and the results tabulated and averaged in Table IV. It shows that flower terminal buds are always associated with comparatively weak growth. Thus sound Seabrook's Black from Chelmsford showed an average length of internode of 2·06 cm. for flower terminals but 3·00 cm. for wood terminals. Long Ashton material gave corresponding figures of 2·03 and 2·42. Reverted Seabrook's Black from Chelmsford off bushes showing no big bud gave approximately equal

figures but nearly all fruit buds, indicating that the bushes as a whole were not growing as strongly as they should be. Sound Boskoops from Chelmsford which were very strong shoots showed no flower terminals at all while Long Ashton material again gave the higher figure for shoots with wood bud terminals. From the evidence therefore it would appear that shoots making a weak growth from whatever cause usually end in a flower bud and such a condition might be expected to cause reverted wood the following season.

Weak growth in any twig may be caused by two conditions. The bush as a whole may be making weak growth or the twig itself may be in such a position as to get a poor sap supply. Suckers from a bush usually make a strong growth the first year, a moderate one the second year and comparatively weak ones in the third and fourth years. In the best pruning method therefore all fourth year wood (if any) and most third year wood is pruned away. Thus none of the weakest wood is allowed to fruit and only part of the rather weak wood, the best and most abundant fruit being obtained from the strong and moderately strong wood. In a healthy bush therefore it is not until the third or fourth year that the terminals are changed into flower buds and so the behaviour of the succeeding year's growth is of no consequence.

Case 4. Aphis attack.

The effect of aphis attack in summer is, as stated above, to cause two or three lateral buds immediately below the point of attack to break into growth. Fig. 14, Pl. VI, shows a typical example and is of course of comparatively common occurrence. True terminal growth has thus been lost and the terminal buds formed on these lateral outgrowths at the end of the season are practically always flower buds. Consequently next year there is a tendency for reverted wood to be formed. Reverted wood means more lateral wood and consequently less sap is available for the terminals. This results again in a tendency to form flower bud terminals and so the process continues. If however the bush as a whole is in very good condition the terminals on the wood formed as a result of the aphis attack may be wood buds in which case there is no immediate tendency to revert. If the growth is not quite so strong but still moderately so the bush may recover its condition in the year following aphis attack and form wood buds as terminals and so escape reversion. The immediate effect therefore of aphis attack will depend on the condition of the bush. It is possible that aphis attack may affect the bush in another way. If as usually happens three shoots appear where in unattacked bushes but one is present it is clear that, if every shoot on a

Fig. 13. Reverted wood of "French" grown at Long Ashton. The strig from last year's terminal flower bud can be clearly seen. The two accessory growing points originally present in the terminal flower bud have in this case both grown, giving the appearance of a divided terminal. Big Bud is present but was not the cause of reversion.

Fig. 14. Growth resulting from aphis check in summer. Specimen from Long Ashton. At the top is the original terminal much weakened by aphis attack. The strength of the shoot, in this case a strong sucker, was thrown almost entirely into the five buds immediately below.

bush behaved so, the total number of terminal growing points or extension shoots would be tripled. If this condition is not remedied at pruning time the bush becomes possessed of far more extension growing points than it can support properly.

Case 5. Through unknown causes.

In addition to the cases already described there is a form of reversion for which at present no explanation can be offered. It occurs in very young bushes that have been cut down immediately after planting and therefore cannot be accounted for by any interference with terminal growth. Instead of making the usual straight growth with broad leaves considerable branching occurs from the laterals made during the current season; at the same time the leaves are markedly pointed in shape. The following year the fruit runs off. This case is being investigated but at present there is no satisfactory explanation.

CONCLUSIONS.

In every case so far examined it has been shown that reverted wood has been caused by a check to the terminal bud. This check may be owing to the normal wood bud terminal being substituted by a big bud, a killed bud, a blind bud or a flower bud. In all cases an abnormal amount of growth from lateral buds situated below, takes place. The exact arrangement of this lateral growth depends on the time and manner of the check to the terminal and has already been discussed. There is at all events little doubt from the evidence given above that such a check does occur and that it suffices to explain the character of reverted wood. The connection between reverted wood and "running off" of the fruit is not quite so clear.

The amount of running off varies with the season. If it is wet the fruit on reverted bushes hangs on very much better than if it is dry, which would point to insufficiency of sap supply as the cause. This is supported by the behaviour of the berries. Usually it is the basal ones that remain but sometimes it is the apical or intermediate ones. In any case they do not drop until about half size, a behaviour which suggests that fertilization has taken place normally. The evolution of the fruit goes on as in a healthy bush until the summer drought begins to be felt and there is nothing to show up to this time that they will not reach maturity. The case may be compared with the June drop of plums where fruits already "set" are dropped in considerable quantities and only the more favourably placed ones are left. If drought in respect of the bush affects the holding power of the fruit it is not unreasonable to look for

some factor causing a local drought to the young fruits. The presence of an unusual number of growing points may supply the answer. They may absorb the sap that should go to the developing fruits and thus cause a local drought. It may be contended that normal bushes often show a considerable amount of branching and yet bear fruit but this case is entirely different. Here the branching is caused by excess of sap from the roots forcing out buds otherwise quiescent, while in truly reverted bushes the sap is diverted from the single terminal growing point into a number of laterals that would in the ordinary course of events have remained quiescent. In the first case there is enough sap for the fruit buds and more to spare for lateral wood growth and in the second a limited supply diverted from the fruits to an unusual number of new growing points. In addition, these extra lateral growths are usually formed comparatively low down on the year's growth and it is possible that as a result they secure the first call on the sap supply. The case may be compared with that of the peach whose method of fruiting is similar. Here also the flowers are borne along the preceding year's shoot and it is a text-book rule to leave a growing point beyond the developing fruits. Failure to do so is said to prevent the fruits from attaining their normal size and in practice often, though not always, does so. In gardening language there is nothing to "draw the sap" to the fruit, in consequence of which the fruits suffer from a local drought. It cannot be too strongly urged that the whole process of reversion is gradual and at no point can hard and fast lines be drawn. This is not surprising when it is considered what complex factors affect the sap supply to the fruits. It will be dependent on the variety, the climate, the condition of the rooting system, the number of active growing points, and so on. One of the most puzzling points about running off is the fact that the sap supply to the fruits would appear to be small at the same time when, to judge from the active growth made by the bush, the sap supply elsewhere is large. Apart from the argument outlined above that the abnormal number of sap drawers deprive the developing fruit of sap there is another point to consider. At the time when the outgrowth of laterals begins the growth push is vigorous (see graphs, p. 15). At the time when the fruit drops, which is somewhat later, the general growth push has slackened though the lateral growths are still actively transpiring and making vigorous calls on the sap supply. Strictly speaking therefore the sap supply to the fruits is small at a time when the sap supply *has been* large to the laterals.

POSSIBLE REMEDIES.

Since there may be several causes of Reversion it is important to determine in any given case which are acting. Reversion caused by the presence of mite can obviously only be cured by the elimination of the mite. There is no satisfactory method yet of doing this though some means of control might be found by using the information afforded by Tables I and II. Investigation will be continued in this direction. Reversion caused by terminal fruit bud formation is evidence either of faulty pruning or of poor condition of the bush as a whole. Correct pruning assisted by judicious manuring or other necessary cultural operations should give a permanent cure. Reversion caused by aphis should be curable by pruning away superabundant terminals; at the same time care should be taken that the bush is making strong growth.

SUMMARY.

1. Reversion is characterised by four symptoms:

(*a*) "running off" of the fruit,

(*b*) unusual amount of lateral wood growth,

(*c*) sharp pointed leaves,

(*d*) long, thin, internodes.

2. It is associated with and apparently caused by a check to the terminal growth through change of the terminal wood bud into:

(*a*) a big bud,

(*b*) a killed bud or blind bud,

(*c*) a fruit bud.

It may also be associated with aphis attack.

3. Mite resistant varieties revert under conditions 2 (*b*) and 2 (*c*).

4. Seabrook's Black is mite resistant because in normal climates and seasons the mite kills the attacked growing point and thus starves itself. This only happens when both climate and variety favour an early and strong continued check to the growth of the bush.

5. There is a form of reversion occurring in young bushes before mite or aphis are present. This form is at present unaccountable.

A "WITHER TIP" OF PLUM TREES

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(With Plates VII—IX.)

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I. GENERAL OBSERVATIONS.

In the summer of 1916, during the course of an investigation into the cause of, and means of controlling, a "Blossom Wilt" of apple trees which proved to be due to *Monilia cinerea* Bon., the attention of the writer was directed to the presence of a *Monilia* on certain withered leaves and dead twigs of Victoria plum trees in the fruit plantation at Wye College. On microscopic examination it was found that the fungus was morphologically similar to that producing the "Blossom Wilt and Canker" disease of apple trees. The colour of the pustules was the same and the dimensions of the conidia when the two forms were found under similar conditions were also in approximate agreement, but since the mode of attack was quite different in the case of plum twigs it was decided that the disease demanded special study.

Observations made on the affected trees showed that leaves actually bearing pustules of the fungus were not of very frequent occurrence, but

there were many leaves which, with the exception that externally no fungus was to be seen on them, resembled those with the *Monilia* pustules on their surface, in that they were dark brown, curled and withered. These diseased leaves were borne on the young green vegetative shoots, *i.e.* the twigs which had developed from buds during that spring. At the time that observations were begun (about the middle of June) the majority of the affected twigs were either wilting at the tip or were quite dead for several inches from the tip downward. Others showed clearly that generally, if not invariably, the damage did not actually commence at the extreme tip of the damaged twig and then extend backward towards the main branch, but that a discoloured (dark brown) area appeared at a node bearing a dead leaf, the node being situated some distance behind the apex. When the discoloured area had girdled the twig the transpiration current became arrested and the terminal portion of the twig thus becoming flaccid began to wilt and the leaves to flag; this flaccidity caused the soft green tip of the shoot to become more or less pendant and the curve produced is generally a characteristic feature of the diseased twigs (see Pl. VII, Fig. 1).

At a later stage the whole of that portion distal to the node where the attack originated is dark brown and withered and the discoloration extends downwards for a short distance below that node, the lower limits of the diseased tissues being distinctly marked off by the contrasting colours of the dead and living parts. As desiccation of the diseased tips becomes more pronounced the affected tissues indurate and the curved condition of the shoots persists through the winter. Since the shoots are often attacked quite early in the season the leaves wither before the absciss layer is developed and so remain on the tree after the normal leaves have fallen. These dead twigs with their persistent withered leaves may easily be recognized during the winter and early spring (Pl. VIII, Fig. 4).

The injury results in the production of gum which in many cases oozes out through the bark and appears at the surface as a rounded drop which hardens on exposure to the air (see Pl. VII, Fig. 2).

Instances were observed where a twig had been attacked through two nodes; thus, in the example shown in Fig. 2 the terminal portion had been killed by a girdling lesion at a short distance behind the apex, below this being a healthy portion followed by another diseased area at a lower node.

In addition to the newly killed shoots the affected trees usually bore a number of dead twigs which, from their situation on the branches,

must have grown out and become infected during the previous year. On the surface of these older dead twigs were numerous *Monilia* pustules and it appeared probable that these had served as sources of infection for the young growth.

When a twig is killed early in the season the buds towards its base which normally would have remained dormant until the following year may be stimulated to precocious development and several weak shoots with pale green leaves grow out. Thus the vigorous leading shoot, which, under ordinary circumstances, was destined to produce a normal supply of young "wood" for the production of fruit buds in the succeeding second or third year, becomes replaced by a number of feeble, immature "unripened" shoots which would probably be killed during a hard winter (Pl. VIII, Fig. 3).

It was noticed that the plum trees that year were suffering from a severe attack by aphides (*Aphis pruni* Réaumur) and these insects were also present in considerable numbers on trees affected by the "Wither Tip" in other plantations in Kent.

Although inoculation experiments carried out at Wye with pure cultures of the fungus have hitherto failed to induce infection of the leaves[1], observations in the open appear to indicate that the young shoots must in many cases (*i.e.* in the typical "Wither Tip" disease) become attacked through infected leaves. Duggar (4) in reference to the "Brown Rot of Stone Fruits" states[2] that "The twigs are also susceptible, but it has been definitely shown that infection of the twigs results only when either flowers or fruit produced on the twigs have already fallen prey to the disease." I have pointed out however (10) that there are reasons for suspecting that the American Brown Rot fungus is a form of *Monilia* quite distinct in some respects from either *M. fructigena* or *M. cinerea* as occurring in this country.

Frank and Krüger (7) have described and figured an instance of infection occurring in the lamina of a cherry leaf, but in this case the leaf was in actual contact with an infected flower. In the "wither-tip" disease of the plum, infection of leaves far removed from any other source of infection is of frequent occurrence.

Aderhold (1) records and figures the wilting of young shoots but only when infected through a node bearing diseased flowers. Frank and Krüger also illustrate a similar instance.

[1] *Vide* p. 38. [2] *Loc. cit.* p. 189.

II. THE FUNGUS.

(*a*) Annual Cycle.

Although the newly killed twigs did not as a rule bear any fungous fructifications except in those comparatively rare instances where *Monilia* pustules appeared on the leaves, the presence of *M. cinerea* on the dead twigs of the previous season suggested that the fungus would appear at the surface of the young affected twigs if these were allowed to remain on the trees until the following year. This seemed all the more probable by analogy with the annual cycle of that form of *Monilia cinerea* which produces the "Blossom Wilt and Canker" disease of apple trees. In a previous paper it has been shown that in the latter the pustules are not generally produced at the surface of the cankers and the dead spurs until the approach of winter, but develop readily from about the beginning of December onward through the winter and spring.

In order to ascertain whether similar conditions obtained in the case of the "Wither Tip" fungus, labels were attached to a number of the dead twigs in the summer of 1916. As was anticipated, grey *Monilia* pustules began to burst through the bark of some of the twigs early in December (a few being found with immature pustules on December 6) and continued to appear throughout the winter. With very few exceptions all the twigs labelled as showing the typical wither tip condition in the summer, bore *Monilia* pustules in winter. Thus on one tree 31 withered tips had been labelled in August 1916. *Monilia* pustules were first observed on December 6 when one of the dead twigs was found bearing the fungus, as pustular tufts of conidiophores, on its surface; later, pustules appeared on others and by May 1917 they were present on all except two of the labelled twigs.

The length of the dead portion had been noted in the case of some of them and in no instance could evidence be found that the fungus had extended any further along the twigs from August 2, 1916, to May 11, 1917. In this again the habit of the fungus found on the plum twigs is comparable with that of the form found on apple spurs and cankers for it has been shown that although the blossom-wilt fungus of apple trees makes rapid progress for a few weeks immediately after infection of the flower occurs its further extension into the tree is inhibited after about the middle of July.

On another tree 25 withered tips had been labelled on August 5, 1916; the *Monilia* appeared at the surface of three of them by December 6 and all bore mature pustules on May 14, 1917, when the tree was in full

bloom. Many of these withered twigs retained their leaves throughout the winter; the presence of such twigs still bearing leaves at that season of the year is an almost certain indication that *Monilia* pustules are to be found.

When the trees were examined in spring a few cankers bearing pustules of the fungus were found on the branches; in each case the canker had arisen round a node, the branch having been invaded through a short shoot or flowering spur.

Since considerable numbers of the dead twigs bearing the fungus were present on the trees under examination it was expected that there would be an outbreak of the disease during the summer of 1917. Very few examples of the typical "Wither Tip" condition were to be found however and it was noticed also that very few aphides were present on the leaves. This suggests that the aphides either aid in the distribution of the conidia or that their presence on the leaves reduces the vitality of the latter to such a degree that they are rendered particularly susceptible to attack by the fungus. The wounds caused by the aphis probably enable the germ tubes to penetrate the epidermal layer.

That outbreaks of the "Wither Tip" disease are dependent on aphis attacks has not yet been established, but the observations made during the past two seasons are suggestive since, although in 1917 (when aphides were few in number) the sources of infection, in the form of withered twigs bearing *Monilia* pustules, were more numerous than in the previous season, instances of twigs killed that season were very few.

(*b*) Cultural Studies.

In order to compare the fungus obtained from the dead twigs with strains of *Monilia* from other sources it was necessary to grow the organism in pure culture, so that its behaviour when growing on varied artificially prepared media could be studied; cultures of the fungus known to be uncontaminated with other organisms were also required for the inoculation experiments described later in this paper. For this purpose infected twigs were obtained from various sources in Kent and in all eleven strains have been grown.

Cultures were obtained initially in two ways, viz.:

(1) If the fungus was producing conidia, as when occurring on the dead twigs in the winter and spring following infection, or as sometimes found on the newly killed leaves in the summer, the conidia were isolated on agar plates and the resulting sporelings which were found to be un-

contaminated were transferred to other plates. When each had reached a sufficient size sub-cultures were made as required.

(2) When the fungus was found as barren mycelium only, as it occurs within the tissues of the newly infected twigs, transverse sections were made through the lower portions of the affected parts, *i.e.* either across the node itself where infection had arisen or through the diseased tissues of the internode below[1]. Particles of the sections were teased out in sterile distilled water and placed on agar plates. The mycelium present in the sections usually grew out uncontaminated by other forms so far as could be seen, but to ensure absolute purity of cultures sub-inoculations were made from the outgrowths by placing peripheral portions of the resulting mycelium on sterilized potato, a medium which has hitherto proved the most favourable for conidia development, and when the conidia were being freely produced some of them were isolated on plates and pure sporelings obtained as described under (1).

Agar-agar, containing an extract of prunes, either as plates or as "slants" in test tubes, was used for studying the mode of growth, and sterilized potato in wide test tubes or in Roux's tubes was used when conidia were required for measuring or for inoculation experiments.

When placed on the nutrient agar the mycelium grows out readily, producing within a few days a regular, almost circular disc of hyphae; later the margin develops into broad lobes with narrow sinuses between them. As in the case of the Apple Blossom-Wilt strains, growth is usually arrested at about 2 cm. from the centre but is resumed as flabelliform outgrowths which generally originate at the sinuses.

It has been shown that the form of *Monilia cinerea* infecting the apple flower, as represented by some thirty strains from various sources, invariably produces a brown coloration when growing on agar-agar containing an extract of prunes. The eleven strains obtained from plum twigs have shown some variation in the intensity of this coloration. One has remained quite colourless in most of the plate cultures of the medium and colourless or almost so as slant cultures in test tubes; one has produced a coloration indistinguishable in intensity from that of the apple blossom strains, while the others are intermediate.

When growing on sterilized potato in test tubes or in Roux's tubes all the "Wither Tip" strains tested have produced a copious supply of conidia, numerous grey tufts of conidiophores, usually in more or less

[1] The terminal internodes, which wither because the transpiration current is interrupted by the death of the tissues actually invaded by the fungus, do not themselves necessarily contain mycelium.

continuous zones, being produced towards the upper end of the potato. In this respect these strains differ from those obtained from the apple since the latter have always produced but a scanty supply of conidia when grown on sterilized potato.

(*c*) Dimensions of the Conidia.

The conidia obtained from a leaf bearing pustules in the summer of 1916 were found to range from $10 \times 8{\cdot}5\mu$ to $23 \times 14{\cdot}5\mu$, the majority however were $16–18 \times 12–14\mu$. In subsequent cases 100 conidia were measured and an average taken. In two instances conidia were obtained in summer from twigs killed during the previous season: the averages for 100 conidia of each were $14{\cdot}0 \times 10{\cdot}5\mu$ and $14{\cdot}0 \times 10{\cdot}0\mu$ respectively. When conidia were taken from dead twigs in the winter or early spring their dimensions were considerably smaller; thus the average of 100 conidia taken from a twig in winter was $11{\cdot}0 \times 8{\cdot}0\mu$, and from another in early spring $10{\cdot}5 \times 7{\cdot}0\mu$, the size varying from $5{\cdot}5 \times 5{\cdot}0\mu$ to $17{\cdot}0 \times 10{\cdot}0\mu$.

By analogy with observations made on *Monilia cinerea* as occurring on the flowering spurs of the apple it was considered that the variation was not due to specific differences in the various strains but was the result of modifications in the substratum (particularly the amount of moisture present), and perhaps also of differences in temperature according to the season during which the conidia were in process of development. To confirm this supposition the various strains were subsequently induced to produce conidia under approximately the same conditions by cultivating them on sterilized potato in the laboratory, and an average was obtained of the dimensions of 100 conidia taken from each culture. The size of the conidia formed under such conditions was found in each strain to approximate to that of conidia taken from infected leaves in summer. One strain, already referred to as producing in early spring conidia averaging $10{\cdot}5 \times 7{\cdot}0\mu$, was also inoculated into plums in the plantation while the fruit were green and still growing: the average size of 100 conidia taken from one plum was $16{\cdot}5 \times 12{\cdot}5\mu$, from another $16{\cdot}0 \times 11{\cdot}0\mu$.

The variation in the dimensions of the conidia of the various strains according to the substratum or to the time of the year is shown here in tabular form.

Table showing variation in the dimensions of conidia of various "Wither Tip" strains of Monilia cinerea[1].

Strain	Source of strain (all obtained from plum trees)	Conidia on twigs killed previous year: Taken in winter or spring	Conidia on twigs killed previous year: Taken in summer	Twig killed during current year: conidia on young leaves	Conidia on potato cultures	Conidia on plums artificially inoculated
a	Dead twig killed previous year	—	14·0 × 10·5 (8 × 6·5—23 × 13)	—	16·5 × 12·0 (8·5 × 7—22 × 15)	—
b	Young twig killed current year	—	—	—	17·0 × 13·5 (10·5 × 9—25 × 18)	—
c	Young twig	—	—	(10 × 8·5—23 × 14·5 but mostly 16—18 × 12—14)	16·0 × 12·5 (9·5 × 8—22 × 18·5)	—
d	Young twig	—	—	—	16·0 × 12·5 (9 × 7—22 × 20)	—
e	Old dead twig	—	14·0 × 10·0 (8·5 × 6·5—25 × 14)	—	16·5 × 12·5 (8·5 × 8—22 × 18)	—
f	Young twig	—	—	—	16·0 × 13·0 (9·5 × 8·5—23 × 18)	—
g	Young twig	—	—	—	16·0 × 12·5 (8 × 7—21 × 19)	—
h	Young twig	—	—	—	18·5 × 14·0 (9·5 × 8—23 × 20)	—
i	Canker on a branch infected through a short shoot	10·5 × 7·0 (6 × 4·5—14 × 9·5)	—	—	16·0 × 11·5 (9·5 × 8—21 × 18)	—
j	Dead twig killed previous year	11·0 × 8·0 (6·5 × 6—17 × 10)	—	—	—	—
k	Dead twig killed previous year	10·5 × 7·0 (5·5 × 5—14·5 × 9)	—	—	16·5 × 12·0 (17·5 × 6—25 × 18·5)	July 18 16·5 × 12·5 (10 × 7·5—27 × 19·5) July 31 (another specimen) 16·0 × 11·0 (10 × 7·5—27 × 14·5)

[1] The readings were taken correct to 0·5 μ and the averages were calculated also correct to the nearest 0·5 μ. The numbers within brackets show the range of variation observed in each case.

(*d*) Identity of the Fungus.

The fructifications found on the dead twigs during winter and spring were those of a *Monilia*; the ashy grey colour of these pustules, their size (not usually exceeding 1 mm. in diameter) and the dimensions of the conidia correspond to descriptions of *M. cinerea* Bon. as given by Aderhold and Ruhland (2) and by Woronin (12). The size of the conidia obtained from *old dead twigs in summer* more nearly coincides with the dimensions given by Aderhold and Ruhland for the conidia of *M. laxa*; strains obtained from such twigs were however indistinguishable in their mode of growth and in the size of their conidia from those strains obtained from twigs producing *in winter* pustules with conidia considerably smaller and comparable with those of *M. cinerea* as given by the same authors, when the various strains are grown as pure cultures under laboratory conditions. It follows that, in considering the dimensions of the conidia as an aid to the identification of species of *Monilia*, the conditions under which the conidia have developed must be taken into account.

Although *Monilia fructigena* occurs frequently on the fruit itself, and in some orchards causes quite as much damage to the ripening and mature plums as *Monilia cinerea*, and although the former may be found commonly on trees affected with the "Wither Tip" disease, the writer has never found *M. fructigena* on the dead twigs nor isolated it from infected young shoots; its occurrence appears to be exclusively on the fruit.

The "Wither Tip" disease of plum trees is therefore to be attributed to *Monilia cinerea* Bon. The writer at present prefers to retain this, the name given to the conidial stage of the fungus, in preference to *Sclerotinia cinerea* for reasons already stated in a previous paper (10).

The "Brown Rot" fungi are known to be responsible for great losses in other parts of the country where the plum is extensively cultivated, particularly in Cambridgeshire, the Evesham and Pershore districts of Worcestershire, and in Devonshire; not only are the fruit, blossom and twigs attacked, but the larger branches are often killed. Since the "Wither Tip" fungus readily attacks both fruit and blossom of the plum tree and may form cankers (as shown in this paper), it appears probable that the same form is the one chiefly responsible for the attacks in the districts mentioned. In Devonshire a disease which appears to be identical with the "Wither Tip" of plum trees in Kent is reported[1] as

[1] *Vide, The Fruit-grower, Fruiterer, Florist and Market Gardener*, June 15, 1916.

causing great damage; it is attributed to *Sclerotinia* (*Monilia*) *fructigena* but is probably caused by *M. cinerea*.

As there is still some confusion in respect to the nomenclature of the Monilias parasitic on fruit trees, and since the work recorded in the present paper affords evidence of the occurrence of biologic forms of *M. cinerea*, it is desirable that a comparison be made of the forms of *Monilia* from various fruit-growing localities; the writer therefore would be glad to receive specimens of affected fruit or branches from other counties that the fungus present on the diseased material may be compared with those strains obtained from trees in Kent.

III. INOCULATION EXPERIMENTS.

During the spring and summer of 1917 inoculation experiments were carried out, using pure cultures of a strain of *Monilia cinerea* obtained from a dead twig bearing *Monilia* pustules, with the object of acquiring further knowledge relative to the mode of parasitism of the fungus, and the extent of the injury caused by it. Several strains had been isolated during the summer of 1916 and grown as pure cultures, but, as some organisms are known to become less virulent after continued growth on artificial media, it was thought desirable to start cultures of a strain isolated during the same season as that in which the inoculations were to be made. Since there was also the possibility of the occurrence of variation in the virulency of different strains of *Monilia cinerea*, particularly as it had been found that cultural variations were to be detected among the strains, it was decided to work throughout with one strain, known to originate in a single conidium and actually obtained from a "withered tip" of a plum tree.

The method adopted in isolating the strain used for the inoculation experiments was as follows:

Towards the end of March 1917, a twig, which had been killed during the previous summer and which bore a number of the grey pustules typical of the fungus, was removed from the tree and placed in a moist chamber. In 48 hours the pustules had become very pulverulent and conidia were removed by means of a sterile needle and placed in sterile distilled water in a watch glass; the conidia floated on the surface and drops of the water were transferred with a platinum wire loop to an agar plate. Some of the drops contained isolated spores and these could be examined under the microscope through the bottom of the plate. After

two days several of the resulting sporelings were seen to be uncontaminated and three were transferred to another plate. All three grew out in a similar manner and were indistinguishable in their mode of growth from other strains of *Monilia cinerea*; sub-cultures from one of these were used in the inoculation experiments. On the agar plates the fungus failed to produce conidia and as these were necessary when infection of flowers *via* the stigma was attempted, cultures growing on sterilized potato were obtained since on this medium conidia readily developed within a week.

(*a*) Inoculation of Plum Leaves.

Since observations in the open suggested that infection of the twigs arose in the leaves, attempts were made to produce the disease by inoculating leaves with the fungus. Experiments were made on the leaves of young growing shoots both in the greenhouse and in the open, conidia being placed (1) directly on the uninjured surface of the leaves, or (2) on the glands at the base of the lamina, or (3) on punctures made through leaves by means of a sterilized needle. In no case however did inoculation result in infection of the leaves and no difference could be detected between inoculated leaves and controls, repeated attempts giving invariably negative results.

It would appear that the leaves must receive some special form of injury before the fungus is able to invade the tissues and the results lend support to the hypothesis that aphides provide the kind of injury necessary for the fungus to establish itself in the leaf.

(*b*) Inoculation of Plum Flowers.

Experiment 1. Inoculation of flowers on a Victoria plum tree in the College Plantation.

A culture of the fungus growing on sterilized potato in a test tube was prepared and when numerous conidia had developed the potato block was transferred from the tube to a sterile petri dish; small portions bearing tufts of conidiophores were cut off with flamed scalpels and transferred to another dish which was then taken to the tree where the experiment was to be carried out. The actual inoculation of a flower was made by removing from the dish one of the particles of potato on the point of a sterilized mounted steel needle, and the conidia-bearing surface was applied to the stigma of the flower.

The tree selected was free from diseased twigs or "mummied" fruit.

Five branches were chosen and in all eighteen umbels of flowers were inoculated: one of these umbels consisted of three flowers, the rest of two flowers each, so that thirty-seven flowers were inoculated. The umbels so treated were mostly at alternate nodes, the intermediate ones being used as controls. Within three days all these flowers showed evidence that infection had occurred, by a brown coloration of the stigmas and in most of them by the discoloration extending also down the style.

At this stage no other flowers on these branches showed any discoloration of the styles or stigmas, thus indicating that the infection was the direct effect of inoculation with the *Monilia* conidia. The results obtained are given below in detail for Branches I and III, on each of which four umbels of two flowers each were inoculated: these, being also typical of the results obtained on the rest, show the progress of the disease after inoculation of the stigmas until the branch itself is invaded.

The inoculations were made on May 11: the nodes bearing the treated flowers were labelled *a*, *b*, *c*, *d*.

Results on the 3rd and 4th days after inoculation.

Branch I.

	MAY 14	MAY 15
(*a*)	Stigma of each of the two flowers brown	Styles brown for 2 and 5 mm. respectively
(*b*)	Stigma of each flower brown: in one the discoloration had extended for 2·5 mm. down the style	Styles brown for 2 and 7 mm. respectively
(*c*)	Stigmas brown: in one the style was also brown for half its length (*i.e.* for about 5 mm.)	Styles brown for 6 and 8 mm. respectively
(*d*)	Stigmas brown: styles also brown for 1 and 5 mm. respectively	Styles brown for 5 and 7 mm. respectively
Controls:	No discoloration, even of the stigmas on the rest of the flowers on the branch	No discoloration of the stigmas

Branch III.

	MAY 14	MAY 15
(*a*)	Both stigmas brown and both styles for 2 mm.	Styles brown: one for 5 mm. the other to the base (about 10 mm.)
(*b*)	Both stigmas brown: styles brown for 3 and 4 mm. respectively	Styles brown for 5 and 7 mm.
(*c*)	Both stigmas brown: styles brown for 2·5 and 4 mm.	One style brown for 6 mm. the other to base
(*d*)	Both stigmas brown; style of one flower brown for 2·5 mm.	Style brown for 5 and 8 mm.
Controls:	No brown stigmas	No brown stigmas

Results on the 6th and 8th days after inoculation.

Branch I.

	May 17	May 19
(*a*)	1. One flower with style brown for 5 mm.	1. Style brown to its base; base of pedicel brown and sulcate for 2 mm.
	2. The other flower with style brown to base; ovary brown, and a brown discoloration is evident on the exterior of the flower at junction of pedicel and calyx tube	2. Whole flower brown and withered, calyx lobes recurved; pedicel brown to base and longitudinally sulcate, discoloration extending upwards into the base of the other flower (No. 1 above)
(*b*)	1. Flower with style brown for 6 mm.	1. Style brown to base
	2. Style brown to base, ovary also brown	2. Nearly the whole of the calyx tube and 2 mm. of the pedicel are brown
(*c*)	1. Style brown to base	1. Style and ovary brown, calyx lobes discoloured and recurved, base of pedicel brown for 5 mm.
	2. Style brown to base, ovary brown, base of calyx tube brown	2. Whole flower withering, calyx lobes recurved, pedicel brown to base and discoloration extending upwards in the other pedicel
(*d*)	1. Style brown to base	1. Style and ovary brown
	2. Style brown to base, ovary and base of calyx tube also brown	2. Whole flower withered and inclined on its pedicel; pedicel brown to base and longitudinally sulcate
	Controls: Of the rest of the flowers on this branch some stigmas show a slight discoloration	Most of the non-inoculated flowers now show a slight discoloration of the stigmas; three also show a browning for 1 mm., while one flower has its style brown to base

Branch III.

	May 17	May 19
(*a*)	Both flowers with brown styles, the discoloration beginning to extend into the ovary	Style and the whole of the ovary brown in both flowers
(*b*)	One flower with style brown for 6 mm. the other with style brown to base	Both styles brown to base; in one flower the discoloration also extends through the ovary and is to be seen on the outside in the basal part of the calyx tube
(*c*)	Both flowers have styles brown to base and the ovaries are also brown; one is also discoloured at the base of the calyx tube	Both flowers brown and withered with recurved sepals; pedicel of one of them brown and sulcate for half its length, the other brown to its base
(*d*)	Each flower has style brown to base and ovary also brown; one has a trace of brown at the base of the calyx tube	Both flowers brown and withered with recurved sepals; pedicel of one brown and sulcate for 1 cm., the other to its base
	Controls: One flower at the node next below (*c*) has its style brown for 1 mm.; otherwise there is no distinct discoloration on any of the non-inoculated flowers of this branch	Some of the non-inoculated flowers still show no discoloration whatever, others have brown stigmas and one has its style brown for 3 mm.

Results on the 10th and 12th days after inoculation.

Branch I.

	MAY 21	MAY 23
(*a*)	1. The discoloration at base of pedicel extends for 1·5 cm. upwards 2. This flower bears small grey *Monilia* pustules	Both flowers are withered to base; the leaves round the base of the umbel are wilting
(*b*)	1. Style, ovary and base of calyx tube brown	1. Flower itself brown; pedicel still green but somewhat sulcate
	2. Pedicel brown for 3 mm. from the calyx downwards; the stamens bear *Monilia* pustules; the flower is detached at its base but is attached to the other flower by its stamens	2. Stamens still entangled with those of No. 1
(*c*)	The pedicels of both flowers are brown to the base and bear *Monilia* pustules	Both flowers quite withered to base; pedicels bear pustules
(*d*)	1. Calyx tube and 1 mm. of pedicel brown	1. Flower brown to base of the pedicel
	2. The pedicel now bears pustules	2. No further evident change
Controls:	The non-inoculated flowers have discoloured stigmas and some have styles brown for about 1 mm., but only one flower is quite dead	The non-inoculated umbels are setting into fruit, except the one at node distal to (*a*) and one other flower which is dead

Branch III.

	MAY 21	MAY 23
(*a*)	Umbel broken off (probably during a thunderstorm of previous evening)	—
(*b*)	1. No further change evident	1. Style brown to base and the discoloration is extending in the upper half of the ovary, which however has increased in size
	2. Calyx tube and half the pedicel brown	2. Broken off
(*c*)	One flower is detached; the pedicel of the other is brown for three-quarters of its length	Flower withered to base of pedicel
(*d*)	Both flowers brown to base of pedicels; one pedicel bears *Monilia* pustules	Both flowers quite withered; the leaves round the base of the umbel are wilting and their petioles are brown at the *base* only (*i.e.* they are being invaded from the point of insertion on the fruiting spur)
Controls:	The non-inoculated flowers generally show a discoloration of the stigmas and a portion of the style (usually about 1 mm.)	The non-inoculated umbels are now setting fruit

Result five weeks after inoculation.

On June 14 the five branches treated showed results of inoculation to be as follows:

Branch I.

This branch was dead at node *c* and from this node distally to the tip of the branch, which has evidently been girdled by a canker at node *c*.

At node *d* the spur was dead but no canker had arisen round its base.

Branch II.

The branch was dead from node *b* to the tip (*i.e.* branch girdled at *b*). Gum was oozing from the dead spur at node *c*.

Branch III.

(*a*) Spur dead but no canker present on the branch.

(*b*) and (*c*). Both spurs alive (*i.e.* leaves showed no signs of wilting) but the flowers had been killed so no fruit had set.

(*d*) Spur dead: there was no evident canker at this node but gum was oozing through the bark just below the node.

Branch IV.

(*a*) Spur dead: a canker half girdled the branch: a large gum globule was present on the branch near the insertion of the spur.

(*b*) The leaves at this node were alive: the dead flowers were still persisting and the spur was gumming round the base of the flowers.

(*c*) Spur dead: a gum globule was present near the insertion of the pedicels: no canker had developed.

Branch V.

This branch was dead from node *c* to the tip: at *d* gum was present and an incipient canker was indicated by a slight depression of the bark on the branch round the base of the spur.

On the rest of the tree at this stage there were only five dead spurs altogether and no branches had been killed except in the case of those produced by artificial inoculation.

General Results of the Inoculation of the Thirty-seven Flowers.

After inoculation the flowers were not examined until the 3rd day; by that time all the stigmas were brown and, with the exception of six flowers, the discoloration had extended from the stigmas down the styles to a distance of 1 to 5 mm. By the following day the browning had proceeded several millimetres further so that in some flowers it had reached the base of the style and in two cases was encroaching on the ovary itself.

On the 6th day the ovary in several of the flowers was brown to the base and the discoloration was also to be seen on the exterior of the flower at the point of insertion of the flower on the pedicel and was extending downwards along the pedicel and upwards towards the tips of the calyx lobes.

8th day: many of the flowers were withered, the calyx lobes being brown and withered, and the pedicels brown and longitudinally sulcate to the point of insertion on the receptacle of the umbel (*i.e.* the tip of the flowering spur); in a few the tissues of the spur itself were invaded as shown by the fact that the discoloration had reached the base of the pedicel of one flower of the pair and was extending *upwards* from the base of the pedicel of the second flower.

At this stage the rest of the flowers on the tree showed generally only a normal discoloration of the stigmas; in a few it had extended for a short distance down the style. Two umbels however showed a browning of style and ovary with the discoloration extending to the pedicels, and it was evident that these had become infected also, the infection probably having arisen by conidia carried by bees from artificially inoculated flowers. There were no flowers quite withered except among those on which conidia had been placed at the beginning of the experiment.

10th day: the disease had extended further into the flowers and pedicels and on some of them *Monilia* pustules with conidia were present. In three cases the fungus had advanced into the tissues of the spurs and by cutting off supplies to the leaves round the bases of the flowers, produced a wilting of the leaves.

12th day: the leaves of other spurs were also wilting and examination showed that the discoloration began at the base of the petiole, thus showing that the disease was invading the leaves from the spur.

With regard to the flowering spurs which had not been artificially inoculated (some hundreds in number) on the same tree, only two at this time bore withered flowers and leaves and one other had dead flowers. These three spurs were all in the neighbourhood of inoculated umbels, being from 9 to 15 inches from the nearest flowers inoculated from the culture; their proximity to the artificially inoculated flowers suggests that bees had carried conidia from the stigmas of the latter to those of neighbouring flowers.

5 weeks: all the inoculated flowers had been dead for some time; at some of the nodes the disease had not only killed the flowering spur but had reached the branch and girdled it (so causing the death of the distal portion) or had produced a canker partly girdling the branch. In some instances gum had issued from the bases of the infected spurs.

It is evident therefore that the strain of *Monilia cinerea* used in this experiment, a strain obtained from a twig affected with the "Wither Tip" disease readily infects plum flowers, invades the spurs, and is able to produce cankers on the branches.

Experiment 2.

While the experiment recorded in the preceding pages was in progress, the stigma of a single flower, one of an umbel consisting of two flowers on a young plum tree growing in a pot in the open, was inoculated for the purpose of obtaining a photographic record of a diseased flower at various stages after infection. Unfortunately a storm removed the flower on the ninth day after inoculation and by that time there were no other flowers available suitable for a repetition of the experiment. However, three photographs were secured and these show the earlier stages of the disease after inoculation of the stigma with conidia of the fungus (Pl. IX, Figs. 7, 8 and 9). The second flower of the umbel served as a control and in the photographs is seen to the left of the infected one.

Inoculation was effected by bringing a particle of potato bearing conidial tufts into contact with the stigma as in Experiment 1, on May 14. The condition of the flower on the days on which the photographs were obtained was as follows:

May 19: style of inoculated flower brown for 3 mm.; the control flower showed no discoloration even of the stigma; the petals of both flowers had fallen by this time (Fig. 7).

May 21: style of inoculated flower brown to base and the discoloration had extended into the ovary (Fig. 8).

May 23: whole of pistil brown, the calyx lobes were becoming withered and recurved: stamens collapsed (Fig. 9).

(*c*) Inoculation of Immature Plums on Trees in the Plantation.

Experiment 1.

The experiment was carried out on young green plums (variety Victoria) at a stage when they were about 2 cm. in length, the date when the inoculations were made being June 20.

Method: conidia were removed with a sterilized scalpel from the surface of a culture on steamed potato and floated on sterile water in a watch-glass which had been previously passed through a flame and allowed to cool. The inoculations consisted in applying with a flamed platinum wire loop drops of the water containing conidia.

The plums were treated in one of three ways:

(1) A drop of the conidia-containing water was applied to the stalk end of the plum (junction of stalk and fruit) without injuring the fruit.

(2) Pairs of plums were selected in which the two of each pair were in contact; one plum of each pair was inoculated by pricking with a sterile needle (a single puncture being made in each case), piercing the skin, and applying a drop of the water with conidia to the wound.

(3) Controls: these plums were each wounded with a single puncture but no conidia were placed on the wound.

The fruiting spurs at which the inoculations were made were labelled and numbered as follows:

1, 2 — Treated as in method (1); both plums at each of the two nodes were inoculated without wounding by conidia in a drop of water placed on the uninjured skin.

3, 4, 5, 6 — At each of these four nodes, one of each pair of plums was inoculated as in method (2); the other plum of each pair was left untreated in any way.

c, *c* — Controls: both plums at each of the two nodes were pierced by a single prick with a sterilized needle but not inoculated.

The above were all on one tree; on the next tree in the row a similar series of inoculations were made, *i.e.*:

7, 8, 9, 10 — Treated as nodes 3–6.

11, 12 — Treated as nodes 1 and 2.

c, *c* — Controls treated as *c*, *c* on other tree.

Thus in all eight plums were treated in each of the three ways.

No rot occurred throughout the experiment on any of those inoculated by placing conidia on the uninjured skin (*i.e.* plums at nodes 1, 2, 11 and 12), nor on those which had been punctured only.

On those inoculated by placing drops of water containing conidia on the punctures the results were as follows:

June 23: no change to be detected in any of them except No. 6 on which a discoloured[1] area extended for 2 to 3 mm. round the wound.

[1] The rot was indicated externally by a dull purple brown discoloration of the skin which was distinctly marked off from the green of the portion to which the rot had not extended.

The progress of the disease to the end of the month is shown in the table:

	June 26	June 28	June 30
3.	Rot extends 2–10 mm. from the wound	Three-quarters of surface discoloured, numerous small grey pustules present	Whole surface discoloured and bearing pustules; skin sulcate
4.	No change	No change	No change
5.	No change	No change	No change
6.	More than half the surface discoloured; numerous minute pustules present	Whole of the surface discoloured and bearing numerous pustules	The other plum of this pair is attached to the inoculated one by a thickened pad of hyphae and a discoloration extends over it for 2–5mm. from the point of contact
7.	Rot extends 4–10 mm. from the wound	Three-quarters of the surface affected; numerous pustules	Both plums had fallen to the ground
8.	About half the surface discoloured; no pustules	Whole surface affected; numerous pustules	Surface sulcate; a hyphal pad attaches the other plum to the affected one, though former shows no rot
9.	No change	No change	No change
10.	Nearly half the surface affected; no pustules	Nearly the whole of the surface is discoloured and bears numerous small pustules	Whole surface affected; no hyphal pad and the other plum is not attached and is not infected

The later stages of the disease in the same plums are shown in the next table:

	July 3	July 11	July 20
3.	Inoculated plum much shrunken and sulcate	Inoculated plum fallen; the other shows no rot	The plum not inoculated still healthy
4.	Drop of gum at wound; slight discoloration	No further change	The gum has dried; black spot 2 mm. diam. round wound
5.	No change	No change	No change
6.	Inoculated plum much shrunken and sulcate; three-quarters of the surface of the second plum is discoloured and bears numerous pustules	Both plums dry and shrivelled; stalk of inoculated plum brown to base, stalk of the other brown nearly to base	Stalks of both brown to base; gum is issuing from the apex of the spur
7.	(Plum fallen)	—	—
8.	Inoculated plum shrunken and sulcate; the other is discoloured over three-quarters of its surface	Both dry and shrivelled; pustules present; stalks brown to base	No further evident change
9.	Drop of gum at wound	No rot	No rot
10.	Inoculated plum shrunken and sulcate; no rot on the other	Hyphal pad present at point of contact of the two plums and the rot is invading the second plum	Both shrunken and wrinkled; surfaces bear numerous pustules; stalks brown to base

Experiment 2.

On June 26, when the plums were about 2·5 cm. long and still quite green, a series of inoculations, similar to that described under Experiment 1, was carried out on two other trees, the only difference being a modification in the manipulation when applying the conidia to the fruit. The inoculations in this instance were made by placing the conidia-bearing surface of a culture on steamed potato (the potato being cut up into small pieces and used as in the experiments on plum flowers) on the fruit; in the case of those plums inoculated through wounds the conidia were brought in contact with the punctured skin and the needle then inserted again in the wound to ensure contact of the conidia with the cells surrounding the wound.

As in the previous experiment no rot occurred except in those plums which had been punctured and conidia placed in the wounds. The modification of the former method of inoculation resulted in more uniform results and each of the eight plums showed distinct evidence on the second day after the treatment that a rot had set in; thus at each of the nodes numbered 3 to 6 the inoculated plums showed a purple discoloration extending for 2 mm. from the wound, while at nodes 7 to 10 the result was similar except that the colour of the affected tissues was not so distinctly marked.

The rapidity with which the rot extended through the infected plums is shown in the accompanying tables. As in Experiment 1, plums at nodes 1, 2, 11 and 12 were inoculated by conidia being placed on the uninjured skin, and again eight plums were punctured but not inoculated; these are not shown in the table since they showed no signs of rot throughout the experiment.

Extent of the affected areas on the 2nd, 4th and 7th days after inoculation made by placing conidia in punctures: the figures indicate how far the discoloration had extended from the wound:

Node	June 28	June 30	July 3
3	2 mm.	9–15 mm.	Whole surface a dull purple brown and bearing pustules
4	2	5–15	Whole surface a dull purple brown and bearing pustules
5	2	8–12	Whole surface a dull purple brown and bearing pustules
6	2	10–15	As in No. 3 but the other plum of the pair is attached by a pad of hyphae
7	2	5–10	Whole surface discoloured and bearing pustules
8	2	8–12	Whole surface discoloured and bearing pustules
9	2	5–10	Whole surface discoloured and bearing pustules
10	2	7–12	As in No. 6

Later results were as follows:

	July 11	July 20
3.	Inoculated plum shrivelled, the other plum attached to it by hyphal pad and discoloured for about one-third of its surface. These two plums were removed and photographed on this date (see Pl. IX, Fig. 5)	
4.	Inoculated plum dry and shrunken, stalk brown to base; the other plum wrinkled, its whole surface discoloured and bearing numerous pustules; browning extending along stalk for 2 mm. from the plum	Both plums quite dry, wrinkled and much shrunken, surface of both bearing numerous pustules with powdery conidia; stalks brown to the base
5.	Inoculated plum with stalk brown to base	No further change evident; no rot on the second plum
6.	Both plums shrunken and wrinkled; stalk of each brown and sulcate	Inoculated plum detached from spur but remains suspended by its attachment to the other
7.	Inoculated plum wrinkled and shrunken, numerous pustules present, stalk brown to base, and detached from the spur but fruit supported by its attachment to the other plum; the latter shrunken, bearing numerous pustules scattered over the discoloured surface, stalk brown to base	No further change noticeable
8.	Inoculated plum shrunken, stalk brown nearly to base, the other plum attached by hyphal pad but not showing any rot	Inoculated plum wrinkled and shrunken, stalk brown to base and detached from spur but plum remains suspended by attachment to the other
9.	Inoculated plum on the ground, surface wrinkled, no rot in second plum	Still no rot in the second plum
10.	Inoculated plum wrinkled and shrunken, stalk brown to base; the second plum is somewhat shrunken, has numerous pustules scattered over the surface, stalk brown nearly to base	Both plums fallen

The experiments prove conclusively that the strain of *Monilia cinerea* obtained from a withered plum twig was able to produce a "brown-rot" of the fruit when the conidia have access to wounds through the skin; conidia placed on the uninjured skin gave negative results in all cases and it would appear therefore that conidia falling on uninjured plums are unable to produce the rot. When however a healthy plum is in contact with an infected one the mycelium in the latter may produce a dense pad of hyphae at the point of contact, thus enabling it to penetrate the skin at that spot and set up a rot in the adjacent plum. When two plums are in contact the cuticle is probably injured by the frequent chafing resulting from the motion of the branch in the wind, for it was noticed that round the point of contact the waxy "bloom" was removed from the skin.

To supplement Experiment 2 another experiment was carried out at the same time in which one plum of a cluster of five was inoculated at a puncture. The disease extended rapidly through the plum primarily infected, then from that to the two in contact with it and from those to the remaining two so that eventually all five were affected. The result as seen on July 21 is shown in Pl. IX, Fig. 6, the plum in the middle being the one inoculated through a puncture on June 26.

It is evident therefore that the twigs affected with the "Wither Tip" disease are a source of danger to the fruit, for although wounds are necessary to enable the fungus to gain an entrance in the first place, observation has shown that injuries severe enough to rupture the skin are not infrequent; bruises and abrasions occur during storms and biting insects pierce the skin and leave open wounds.

(*d*) Inoculation of Apple Flowers.

Since the strain of *Monilia cinerea* obtained from the plum twig and used in the experiments described in the preceding pages was morphologically similar to the form which produces the "Blossom Wilt and Canker" disease of apple trees it was to be expected that it too was capable of producing the "Blossom Wilt" when inoculated into apple flowers, although it is to be observed that the particular strain used in the experiments on plum flowers and fruit could be distinguished *in culture* from that which so readily infects the flowers of the apple, in that the former remains hyaline[1] in agar cultures containing prune juice while the latter always produces a dark brown coloration when growing on that medium. Experiments were therefore carried out to ascertain whether this hyaline strain from the plum could attack the flowers and spurs of the apple.

Experiment 1.

Young trees of the Worcester Pearmain variety growing in pots were used. A similar experiment on this variety carried out in 1917 but using a strain obtained from a *Monilia* canker of an apple tree resulted in the typical blossom wilt condition in two out of six flowers inoculated. In the present experiment, using the hyaline strain employed in inoculating the plum flowers and fruit, seven flowers were inoculated, viz., the central flower of each of seven umbels. Newly opened flowers (*i.e.* those

[1] One tube culture of this strain did give rise to irregularly distributed brownish patches but even then it was easily distinguished from the much darker cultures of the various strains obtained from apple trees.

which had expanded their petals during the preceding 24 hours) were selected, for in the experiments of the previous years it had been found that such flowers had been more readily attacked than older flowers.

Three of the flowers (numbered 1, 2 and 3) were inoculated on May 14; two others of the same age were left untreated and labelled as controls (c_1 and c_2). By the following day other flowers had opened and four of these were inoculated (numbered 4, 5, 6 and 7); three similar ones were kept as controls (c_3, c_4 and c_5). No difficulty was experienced in obtaining conidia for the inoculations as this strain of *Monilia* produces numerous conidia on sterilized potato.

The result is shown in the table (p. 51).

The early inception of a brown discoloration of the inoculated flowers afforded evidence that infection had occurred. Later, all the inoculated flowers fell away and in no case was there any evidence that the disease had passed from the infected flowers into the tissues of the spur, and the leaves borne on these spurs remained green and healthy. It would appear therefore that the strain of *Monilia cinerea* used in this experiment is able to infect and cause the death of apple flowers but is unable to penetrate further than the base of the pedicels.

This result was particularly interesting as it suggested that there were strains within the same morphological species, *Monilia cinerea*, with different powers of infection. For confirmation of this it was decided to repeat the experiment side by side with one in which a strain from an apple tree was used. To obtain results strictly comparable one tree was selected, one of the Warner's King variety growing in the College plantation; umbels on one side of the tree were inoculated with the plum strain, those on the other with the apple strain. Each of the two strains had been isolated during the previous winter so that there was no question that either had lost its virulency from long continued growth on artificially prepared culture media and it had been already ascertained that the plum strain readily attacked plum flowers and spurs.

Experiment 2.

Method. Particles bearing conidial tufts were cut from a culture of the "Wither Tip" strain growing on sterilized potato. These particles were placed in a sterile petri dish and taken to the plantation, where, by means of needles (previously flamed), they were inserted in the flowers to be inoculated in such a way that the conidiophores were brought in contact with the stigmas. That many of the conidia adhered was evident,

Table showing result after inoculating apple flowers with a strain of Monilia cinerea *obtained from a withered plum twig.*

1, 2, 3, inoculated May 14: controls c_1 and c_2.
4, 5, 6, 7, inoculated May 15: controls c_3, c_4 and c_5.

	MAY 19	MAY 21	MAY 24	MAY 29
1	Styles brown for half their length (about 4 mm.)	Styles brown to base	Stamens collapsed; calyx lobes becoming desiccated and brown	Flower had become detached
2	Styles brown for 4 mm.	Styles brown to base	Some of the stamens collapsed; calyx lobes becoming desiccated	Calyx and stamens withered
3	Styles brown for 1–2 mm.	Styles brown to base	Stamens collapsed; calyx lobes becoming desiccated	Flower had become detached
c_1	Slight discoloration of stigmas only	Stigmas brown: one style with slight discoloration	Styles brown for about half their length; stamens upright	Calyx and stamens withered
c_2	No discoloration even of the stigmas	Stigmas and one style brown	Styles brown for about half their length; stamens upright	Calyx and stamens withered
4	No discoloration	Stigmas brown	Styles brown for about half their length	Calyx and stamens withered
5	Distinct browning of the stigmas and of one style for 1 mm.	Styles brown to base	Styles brown to base; some of the stamens collapsed	Calyx and stamens withered
6	Distinct browning of the stigmas	Styles brown to base	Styles brown to base; stamens all collapsed except two	Calyx and stamens withered
7	Stigmas brown	Stigmas brown	Styles brown to base; stamens spreading	Calyx and stamens withered
c_3	No discoloration even of the stigmas	Slight discoloration of the stigmas	Stigmas brown; slight browning of styles; stamens upright	Calyx and stamens withered
c_3	No discoloration even of the stigmas	Slight discoloration of the stigmas	Stigmas brown; slight browning of styles; stamens upright	Calyx and stamens withered
c_4	No discoloration even of the stigmas	Slight discoloration of the stigmas	Styles brown for half their length; stamens upright	Calyx and stamens withered

for in some cases it was seen that the stigmas were greyish after the application.

Four umbels were so treated, the central flower (which opens first) and one or two others in each umbel being inoculated. The spurs bearing these umbels were numbered and labelled, and notes were made respecting the situation of the inoculated flowers in the umbels.

Umbel No. 1. Three flowers inoculated.
,, *No.* 2. Two ,, ,,
,, *No.* 3. Three ,, ,,
,, *No.* 4. Two ,, ,,

Thus in this experiment ten flowers were in all inoculated with the "Wither Tip" strain of *Monilia*.

In order to avoid any inter-contamination between the two strains, the particles of potato were replaced in the dish and taken back to the laboratory. The needles were re-flamed and particles of a potato culture of the "Blossom Wilt" strain of *Monilia* from apple trees were then prepared and inoculations made on flowers on the opposite side of the same tree. In this case only one flower of each of four umbels was inoculated.

The inoculations were made on May 17 and the results as observed at intervals of a few days are here shown for comparison:

Apple Flowers inoculated May 17.

"Wither Tip" strain obtained from a plum twig	"Blossom Wilt" strain obtained from an apple tree
May 20	
All the inoculated flowers had brown stigmas; some of the styles were also discoloured for 1 mm.	All the inoculated flowers had brown stigmas and the styles were brown for 1–3 mm.
The rest of the flowers of the same umbels and those of other umbels in the vicinity showed no discoloration whatever	No other flowers on these branches showed any discoloration
May 23	
The styles of the inoculated flowers were brown for from one-half to the whole of their length.	The styles of all the four inoculated flowers were brown to the base.
Most of the untreated flowers showed no discoloration at all; in a few cases a slight browning of the stigmas was seen and one style was brown for about half its length	Uninoculated flowers on the same side of the tree showed a slight discoloration of the stigmas only
May 26	
Umbel No. 1. Styles of all three inoculated flowers brown to base; in two of the flowers the discoloration was also extending into the ovary and the stamens were drooping or quite collapsed	*No.* 1. Inoculated flower was brown with collapsed stamens, pedicel brown and shrunken (longitudinally sulcate), bases of filaments with pustules of the fungus

May 26 (*cont.*)

No. 2. The styles of one flower were brown almost to the base; in the other flower the styles were brown throughout their whole length

No. 3. All styles of the three flowers brown to base; stamens upright

No. 4. Styles of both flowers brown to base; stamens upright. Uninoculated flowers had, in general, brown stigmas only but in some the discoloration extended half-way down the styles; no drooping stamens observed

No. 2. Flower with stamens collapsed, calyx lobes withering, leaves round base of umbel beginning to wilt

No. 3. Stamens collapsed; calyx lobes withering; leaves show a slight wilting

No. 4. Stamens collapsed, but no wilting of leaves noticeable. Control flowers of the same age in the vicinity had styles brown for about half their length, there was no withering of calyx lobes and the stamens were all upright

MAY 29

No. 1. Two of the flowers had fallen away, the third had remained apparently unchanged since the 26th

No. 2. Styles brown to base, and in one flower the calyx lobes were withered

No. 3. No further change noticeable except that in one flower the stamens were drooping

No. 4. No further change noticeable. Many of the control flowers by this time had styles brown to base; stamens generally upright; calyx lobes not withered

In all four the infection had by this date extended from the inoculated flower into the tissues of the spur as shown by the fact that in each case the leaves round the base of the umbel of flowers showed a distinct wilting, were turning brown and their margins were more or less curved inward

JUNE 6

There was no wilting of the leaves on any of the four spurs; the inoculated flowers had all failed to set into fruit, they had become withered and *fallen off*; of the uninoculated flowers some were setting into fruit:

No. 1. One flower setting into fruit

No. 2. None setting

No. 3. Two flowers setting

No. 4. Two flowers setting

The young fruit on these spurs were 1–1·5 cm. in length

JUNE 4

All the flowers (those untreated as well as those inoculated) and the leaves of the four spurs were quite withered; the dead flowers *remained in position* on the spurs, this condition being typical of the "Blossom Wilt" disease of the apple tree

No. 4 was removed and mycelium was found in the tissues of the spur

JUNE 7

The other three spurs were removed and cut longitudinally; in each case the young growth of the spur (bearing the leaves and flowers) was quite brown throughout and the browning had extended along the bark of the older portion for a distance of about 3 cm.

(*e*) DEDUCTIONS FROM RESULTS OF INOCULATION EXPERIMENTS.

The experiments show that the particular strain of *Monilia cinerea* obtained from the plum twig is less virulent as a parasite on the apple than the apple blossom strain itself since it was unable to establish itself in the spurs of the trees inoculated, and this in spite of the fact that the method of inoculation adopted favoured the "Wither Tip" strain; thus in the second experiment ten flowers were inoculated with this strain as

against four treated with the apple "blossom-wilt" form, and the conidia of the culture used in the inoculations were more abundant, so there could be no doubt therefore that conidia were successfully applied to the stigmas.

The progress of the disease was almost the same for both (the "blossom-wilt" strain advanced a little more rapidly than the "wither tip" strain) until the base of the pedicel was reached, then in the one case the infected flower fell off without causing any further injury, in the other the flower not only remained in position but the disease extended from it into the tissues of the spur and affected the whole cluster of flowers and leaves.

In this connection it is to be remarked that Woronin(12) in his inoculation experiments with *Monilia cinerea* and *M. fructigena* was unable to produce with *M. cinerea* any infection of apple flowers except on the stigmas themselves. Thus he states[1]: "Die Gonidien der *Sclerotinia cinerea* wachsen auch unter der Einwirkung des Narbensaftes in Keimfäden aus, dieselben können aber nur den Griffel etwas angreifen und sind gar nicht im Stande weiter in die Apfelblüthe einzudringen." It is probable therefore that Woronin worked with a strain of *M. cinerea* obtained from either cherry or plum, since I have had no difficulty whatever in infecting flowering spurs and branches of apple trees by inoculating the stigmas of the flowers with a strain of *M. cinerea* from an apple tree.

Whether other strains of *Monilia cinerea* found on "stone-fruit" trees behave as the "wither tip" strain used in the experiments has not yet been determined and it is proposed to carry out other experiments to ascertain whether there are strains occurring on plums and cherries capable of infecting the spurs of the apple through the flowers. The economic aspect of this problem is involved in the fact that plum trees are often interplanted with apple trees in mixed orchards and the possibility of the former proving sources of infection for the latter or *vice versa* is a point of importance to the fruit grower.

As already stated the "wither tip" strain used was one which usually remains quite hyaline when growing on prune-juice agar; others which have been isolated have more nearly approached the apple "blossom-wilt" form in that they produce a brown coloration when growing on that medium; one in particular (strain *g* of the table on p. 35) is in this respect practically indistinguishable from the apple strains. The "wither tip" strains which have been isolated and cultivated all produce

[1] *Loc. cit.* p. 25.

conidia readily when growing on semi-cylinders of sterilized potato in Roux' tubes, the tufts of conidiophores being produced in more or less continuous dark-grey parallel zones on the upper (thinner) half of the potato. The apple "blossom-wilt" strains on the other hand have always produced but a scanty crop of conidia when growing on potato, the conidiophores developing in relatively few isolated tufts.

The results afford evidence of the occurrence of biologic forms within the species *Monilia cinerea*. That forms morphologically similar but distinct in their biological relations with the host plant, occur among the obligate parasites, the *Uredineae* and the *Erysiphaceae*, has been fully proved (9) and there is evidence that such forms occur also in other groups; thus Grossenbacher and Duggar (8) conclude that *Botryosphaeria ribis* occurs as two forms, one parasitic on currant shoots, the other apparently saprophytic only and they find that the two forms can be distinguished by cultural characters.

In *Monilia cinerea* the two strains which have behaved as biologic forms can also be distinguished culturally and this suggests that biochemical methods may throw some light on the physiological relations of biologic forms in those fungi which, like *Botryosphaeria ribis* and *Monilia* spp., allow of culture on artificially prepared media. Alsberg and Black (3) working with several species of Penicillium pointed out in 1913 the desirability of applying biochemical methods in distinguishing the lower forms of fungi which are not readily to be distinguished by their morphological characters.

In the case of the two strains of *Monilia cinerea*, the difference in coloration suggested a differential action of some oxidising enzyme and preliminary experiments have shown that one strain produces a substance of an enzymic nature, probably an oxidase, much more readily than the other. These experiments are being continued for these two strains and will be amplified by application to other strains.

IV. CONTROL MEASURES.

From the inoculation experiments recorded in this paper it is evident that the form of *Monilia cinerea* which occurs on the dead twigs of plum trees is capable of infecting the flowers and causing a "brown rot" of the fruit of the plum tree, and that such twigs if not removed serve as sources of infection since they produce pulverulent pustules of conidia by the time the trees are in flower. Although inoculation of the leaves with pure cultures of the fungus have, up to the present, given negative

results it would seem that the young leaves also become infected from the dead shoots under certain conditions not yet understood. In severe attacks many of the leading shoots are killed and fruit-buds are replaced by the weak "unripe" twigs, that are induced to grow out. The diseased twigs should therefore wherever possible be removed from the trees together with all rotting and mummied fruit.

Some growers, at the time the fruit is gathered, encourage their pickers to remove all the plums affected with "brown rot." If this system became more general and could be extended to the pinching off of all "withered tips" also, the amount of fruit destroyed each year by "brown rot" would be reduced considerably. As an alternative the dead twigs could be removed in winter when, as a rule, they are easily distinguished by the recurved apex and by the persistent withered leaves. Where practicable they should be removed and burnt but in any case they are far less dangerous on the ground than if left on the tree. To test this point thirty-nine withered tips were removed from three trees and placed on the ground under the trees in September 1916; when examined in the spring of 1917 five of them only had pustules of the fungus, whereas of those left on the trees all, with very few exceptions, produced pustules.

As in the case of the "Blossom Wilt" disease of apple trees spraying with ordinary fungicides is not likely to prove nearly so efficacious as the removal of the dead twigs and diseased fruit. The stigmas of the open flowers are liable to become infected and these cannot be covered with a spray fluid without great risk of injury to the blossom. The fruit becomes infected through wounds and therefore again spraying cannot prove sufficiently protective. A spray that would kill the pustules or render them sterile might be effective but since the diseased twigs are very slender and often at the extreme tips of the branches great care would be necessary during the application to cover them with the spray and large quantities of the fluid would be wasted.

Experiments however were carried out to ascertain the effect of fungicides on the pustules borne by diseased twigs, attention being confined to preventing the pustules from shedding their conidia during that period of the year when new infection occurred. Twigs bearing pulverulent pustules were taken from the trees and atomized with the various spray fluids, then placed in the open, that they might be subjected to ordinary atmospheric conditions, and examined from time to time.

It was found that the commonly employed fungicides such as Bordeaux Mixture, soda Bordeaux ("Burgundy" Mixture), cupram, eau

celeste and lime-sulphur, were almost useless in this respect since, as a rule, they failed even to wet the pustules. They became more effective however when prepared so as to contain 1 per cent. soft soap[1] for then the pustules were wetted and the surface layer of conidia was killed. Ammonium sulphide with soft soap, as recommended by Eyre and Salmon (5, 6) for use against the oidium stage of the *Erysiphaceae*, was also tried with similar result. The pustules were thus prevented from shedding conidia for some time but they were not killed and during a period of wet weather the pustules evinced a tendency to grow out through the superficial barren layer and once more become powdery. Under favourable dry atmospheric conditions therefore such fungicides, when used with soap to reduce their surface tension and increase their wetting properties, would tend to check the spread of the disease if applied just before the flower buds open, to render the pustules non-infective during the flowering period, and again when the fruit has set to prevent the dispersion of the conidia while the fruit is growing. They are not to be relied on however from the fact that the pustules themselves remain alive and re-develop, producing fresh crops of conidia, during periods of moist weather.

Better results were obtained with a solution consisting of 1 per cent. caustic soda and 1 per cent. soft soap, the pustules remaining dark brown for fully three weeks after the application of the mixture; this solution would in practice be used as a "winter-wash" as its caustic properties would cause serious damage to growing shoots. Experiments with this spray-fluid have not been carried out in the open on plum trees, but results of trials on apple trees bearing similar pustules of the "Blossom Wilt" *Monilia* were not such as to warrant its recommendation.

V. SUMMARY AND CONCLUSIONS.

1. In the summer of 1916 many plum trees in Kent were affected with a disease which caused the wilting and death of the tips of young twigs.

2. On each diseased shoot one or two leaves became infected and the disease extended into the tissues of the shoot causing the wilting and death of that portion distal to the node bearing the withered leaf.

3. Such twigs bore pustules of *Monilia cinerea* Bon. during the following winter and spring.

[1] It was found that soft soap could be successfully mixed with each of the sprays mentioned except lime-sulphur.

4. Inoculation of plum leaves with pure cultures of a strain of the fungus obtained from a withered twig gave negative results.

5. Inoculation of the stigmas of plum flowers resulted in every case in infection and consequent death of the flowers; in some instances the infection not only killed the flowering spur but invaded the branch and formed a canker.

6. Inoculation of the fruit gave negative results on uninjured plums, but when the conidia were applied to wounds they produced a "brown rot" which rapidly extended through the fruit and infected other plums which were in contact with those primarily inoculated.

7. Inoculation of apple flowers with conidia of the "wither tip" strain resulted in the death of the inoculated flowers only; these fell off and infection extended no further. On the other hand inoculation of apple flowers with a "blossom wilt" strain from an apple tree not only caused infection of the treated flowers but the fungus extended also into the tissues of the spurs and killed all the flowers and leaves borne by these spurs.

8. The two strains of *Monilia cinerea* used in these experiments are indistinguishable morphologically and, since they are dissimilar in their powers of causing infection of the apple blossom, are to be considered as "biologic forms" or physiological strains. They can be distinguished by applying cultural and biochemical methods.

9. Spraying as a means of controlling the "Wither Tip" disease is not to be recommended. All diseased twigs and fruit affected with "brown rot" should be removed from the trees, otherwise they will prove sources of infection for the flowers and fruit of the following season.

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Fig. 1

Fig. 2

Fig. 4

Fig. 3

Fig. 8

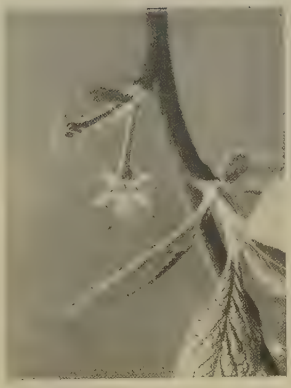

Fig. 9

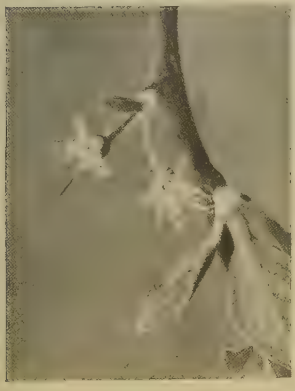

Fig. 7

Fig. 6

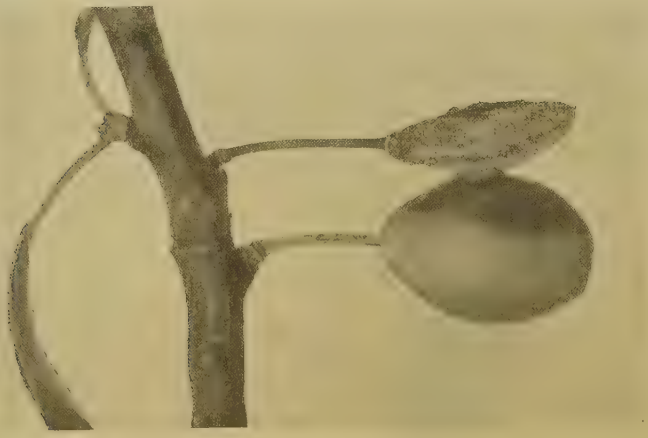

Fig. 5

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DESCRIPTION OF PLATES VII—IX.

Fig. 1. A typical instance of "Wither Tip." Infection arose in the leaf on the left and, extending into the shoot, caused the wilting of the terminal portion.

Fig. 2. In this case infection had occurred in two places; the apical portion is withered from infection through one of the upper leaves; a later attack originated in the second leaf from below, where a drop of gum is seen at the base of the affected leaf.

Fig. 3. The death of the leading shoot has resulted in the outgrowth of two weak shoots with small pale leaves.

Fig. 4. The "withered tips" as seen in winter and spring (photographed March 18, 1917); remains of dead leaves still present and Monilia pustules have appeared.

Fig. 5. The plum on the right was inoculated on June 26 with conidia of a "wither tip" strain of *Monilia cinerea*. Result on July 11—the inoculated plum is covered with pustules of the fungus and the rot has extended into the other plum in contact with it; the pad of mycelium connecting the two is seen.

Fig. 6. A group of five plums with *Monilia cinerea*. One of these plums was inoculated on June 26; the rot gradually extended through it and infected the rest. Result on July 21—all were infected and bore pustules of the fungus.

Fig. 7. The upper flower was inoculated with conidia on May 14. Result on May 19—style discoloured for 3 mm.

Fig. 8. The same on May 21: style brown to base.

Fig. 9. The same on May 23: calyx lobes withered and recurved; stamens collapsed

OVIPOSITION IN THE CELERY FLY.

By T. H. TAYLOR.

(With one Text-figure.)

The habits of the Celery Fly are comparatively easy to study, for, besides being conspicuously coloured and of fairly large size, the flies are obliging enough to proceed with their occupations when the observer approaches, gaily sporting their dappled wings and seeming but little embarrassed by his presence. A sunny afternoon is a favourable time for watching the flies lay their eggs, and although the details of the process are too minute to be seen from a distance, its general features can be followed without much trouble. After sunning itself on the upper surface of a leaf, a fly about to lay, crawls over the edge to the underside of the leaf, and, testing the surface with its tongue, proceeds to drive the tip of the ovipositor into the interior of the leaf. In a few seconds, the boring action ceases and is shortly followed by more generalised movements of the body such as in an insect are commonly associated with the process of egg-laying, and whose purpose is clearly to disengage an egg and pass it to the outside. Visiting other and often adjacent parts of the leaf, the insect lays two or three eggs one after another in quick succession, and then without displaying any further interest in the results of its labour flies off to fresh ground. If the leaf be now plucked and the lower surface examined with a lens, the punctures made by the ovipositor can be seen and also the eggs themselves each lying beneath the cuticle in a separate space in the parenchyma.

By imprisoning a fly in a glass tube or capsule and giving it a piece of celery leaf, one can watch the process of egg-laying under the microscope. After the pointed end of the ovipositor has pierced the epidermis, the body of the tube, two-edged and file-like, forming an efficient boring apparatus, passes into the tissue beneath and breaking down the cells around it prepares a cavity for the reception of the egg. Having cleared a suitable space, the fly charges the tube which, still extended into the leaf, has become for the moment relaxed and inert, and then, with a final effort expels the egg through the terminal opening and crawls away. The process from the insertion of the tube to its withdrawal lasts from about

35 to 50 seconds, the duration being influenced no doubt by the species and age of the leaf, the vigour of the fly and other factors. In captivity, a fly sometimes oviposits in a leaf while crawling on the upper surface, and on these occasions, probably because the tissue is more compact on this side, the process lasts somewhat longer. For example, it was noticed that a fly which spent about 50 seconds in passing an egg into a piece of celery leaf from above, required about 10 seconds less when inserting an egg from below.

At hatching, which takes place in about six days, the larva breaks through the egg-shell at the end pointing away from the puncture in the cuticle, and burrowing forwards, eats out at first a narrow gallery in the tissue, but after a few days changes its mode of attack, and mining in all directions gives rise to a wider cavity less regular in shape. Increasing in size as the larva feeds, the cavity thus produced often coalesces with others of the same kind, and, with these additions, forms a compound blister containing it may be several larvae—a circumstance which has perhaps given rise to the existing impression that the Celery Fly, like Pegomyia and other blister-making flies, lays its eggs upon the surface, whereas it resembles more closely such flies as the Phytomyzidae, which lay their eggs in the interior of leaves.

Celery-fly Egg in leaf of Heracleum, showing puncture made by fly.
(Drawn by Miss E. M. Wright.)

AN EPITOME OF BACTERIAL DISEASES OF PLANTS IN GREAT BRITAIN AND IRELAND.

By SYDNEY G. PAINE.

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The study of bacteria in relation to plant diseases is a branch of plant pathology which has been largely neglected in this country. In fact it is unfortunate that plant pathology as a whole has not received the amount of attention at the hands of English botanists that its importance in agriculture and horticulture demands. Bacteriology in particular has suffered from neglect to such an extent that the knowledge of bacterial diseases of plants occurring in this country is limited to a very few diseases only. It would indeed be fortunate if this were due to the non-existence of others, but there is no reason to believe that the activities of bacterial parasites are less here than they are in other countries with a temperate climate. The unsatisfactory state of affairs was deplored by Prof. Potter in 1910(35) and since that time very little has been done to improve the position. The question calls for the urgent attention of botanists in order that this, which can only be described as a national disgrace, may be immediately remedied. Until quite recently no one in this country has applied himself solely to the study of bacterial diseases.

The leaflets of the Board of Agriculture include only six diseases attributed to bacteria(25) whereas in a general conspectus of bacterial diseases E. F. Smith(45) has compiled a list of 140 genera distributed through more than fifty families in which such diseases are known to occur.

Our present knowledge of bacterial diseases is mainly due to the American plant pathologists and the main reason why we in this country are so far behind the Americans in this respect is that we have no state-aided department of plant pathology which is in the least comparable with that of the American Bureau of Plant Industry. Other reasons are to be found in the facts that plant pathology and plant bacteriology in particular as a general rule form either no part or only a very subsidiary part of the curriculum of the botany student and that where such training is included in his curriculum the student is more attracted by the

mycological than by the bacteriological side of plant pathology. The investigation of bacterial parasitism in plants above all things demands of the plant pathologist an extremity of patience with which few perhaps are gifted. The difficulties met with in the isolation of the parasite from diseased plant tissues are infinitely greater than those encountered in the isolation of an animal parasite. The animal pathologist has in the blood a very specialised medium which excludes almost entirely the invasion of saprophytic forms; on the contrary, the sap of the plant cells is eminently suitable as a medium for the growth of many common bacteria, and, situated as the plant is in a soil containing upwards of five million organisms per gram, when once its defence against parasitism has been broken down by the entry of a parasite, many of these common soil organisms enter the diseased tissue and multiply luxuriantly often to the partial, and sometimes even to the total, exclusion of the original parasite. In attempts to isolate the parasite from potato stems affected with "Blackleg" the author spent several months battling with a multitude of saprophytes before success attended his efforts, and then only by chance was a colony of the parasite discovered on a plate containing some 200 others of which thirty had been already investigated and proved to be saprophytic organisms. In one investigation upon which he is at present engaged more than 400 cultures have already been made and many more will be necessary before the investigation is satisfactorily concluded. Again Smith and Townsend(44) worked most patiently for three years upon the "Crown Gall" disease before the causal organism was tracked to his lair. Another instance may be cited in emphasis of the difficulties encountered in this branch of pathology; Schiff-Giorgini(41) in investigating the Olive Knot disease worked for two years upon an organism thought to be *B. Oleae* which was later shown by Smith(43) to be a common soil organism and incapable of producing the disease. To quote from Smith(45), "The labor involved is enormous and exacting to discouragement at times, the results come so slowly, so much must be done to be certain of so little, all because the organisms dealt with are very small—*how small*, we seldom realise!"

The British literature on bacterial diseases is so scattered and so fragmentary that it seems advisable to collect together the fragments and to "take stock" of our position. An attempt is therefore made in this article to bring together the known facts about our bacterial diseases in the hope that it may be useful to those engaged in diagnostic work, and that it may in some measure stimulate the advancement of this important branch of phytopathology.

White Rot of Turnips and other Vegetables.

This disease investigated by Prof. Potter in 1900(36) was the first disease of bacterial origin to be recognised in this country. It is characterised by the complete rotting of the tissue attacked, the bacteria secreting an enzyme which has the power of dissolving away the middle lamella from between the cells thus destroying the coherence of the tissue and giving rise to the condition of a "Soft rot."

Symptoms of the disease. The first symptoms of disease appear in the autumn as a wilting and yellowing of the oldest leaves which droop to the ground, shrivel up and finally rot away. During a fortnight or three weeks the signs of disease progress inwards from the oldest to the youngest leaves until at the end of this period the plant has completely gone to the ground as a rotting, stinking mass. The root is found at this time to be partially or wholly reduced to a greyish white or brown pulpy mass.

The parasite. Potter was the first to isolate an organism in connection with this disease. He described a small uniflagellate rod under the name *Pseudomonas destructans*. Johnson and Adams(19) isolated an organism from rotted turnips in the West of Ireland which they believed to be Potter's organism, and also found it to be uniflagellate. They, however, draw attention to the close similarity of this to Bacillus Oleraceae isolated by Harrison(16). Jones(22) obtained a culture of a second isolation from Potter and found the organism to be a bacillus, and Harding and Morse(22) have shown that under similar cultural conditions both Harrison's organism and Potter's second isolation are identical with *B. carotovorus* Jones. The writer obtained in 1912 a culture of *P. destructans* from Kràl's laboratory and a slide stained by Loeffler's method showed flagella very indistinctly but each organism appeared to have a single flagellum. In 1916 it was restained by Steven's modification of Van Ermengen's stain and it was then clearly seen that the majority of the organisms were uniflagellate but that some, perhaps 10 per cent., were bacilli. That the number of flagella in bacteria is somewhat variable has been pointed out by Johnson and Adams(19) and the writer has found that *B. atrosepticus* grown on living turnips, and also sometimes on living potato, is present mainly in a uniflagellate condition. Potter has agreed(37) that his organism may have been a bacillus. Upon the evidence, therefore, it seems well to abolish the name *P. destructans* until a definitely uniflagellate organism answering exactly to the description given by Potter shall be again obtained from rotted turnips or other tissue.

The causal organism is then *B. carotovorus*, a rod-shaped organism of varying length motile by 3–6 peritrichous flagella. It stains well with the usual stains and is Gram negative[1].

Host plants and distribution. The distribution of the disease seems to be general throughout Great Britain and Ireland and a great variety of hosts are liable to attack. *B. carotovorus* has been found by several investigators to be capable of producing rot in a great variety of plants, in the roots of carrot, white turnip, parsnip, rape, radish, salsify, in onion bulbs, hyacinth corms, cabbage, cauliflower, celery stalks and potato tubers. No rot has been produced in young carrots or parsnips, apple, pear, beetroot, sugar beet, swede turnips or tomato stems.

Heart-rot of Celery.

A soft rot of celery, in which sometimes the petioles of the outer leaves only, but in more serious cases the heart and subsequently the whole plant, are reduced to a yellowish brown decaying mass, was described by Wormald in 1914 (49) and an organism was isolated and named *B. apiovorus*. The disease had previously been observed in Ireland by Johnson and Adams in 1910 (19) who had isolated an organism but had not studied it fully. In a later paper Wormald (50) gives the results of a comparison of his organism with a strain of *B. carotovorus*, which we have had growing in this laboratory since 1912 when it was obtained from America through the courtesy of Dr E. F. Smith. As the result of this later work Wormald concludes that the two organisms are identical and that "Heart-rot" of celery is the work of that omnivorous organism *B. carotovorus*.

"Blackleg" of the Potato.

This is a disease which produces entire destruction of the plant when the attack occurs early in the season and a disastrous soft rot of the tubers during storage when, following a late attack in the field, a certain percentage of the tubers are so slightly affected at the time of lifting that their diseased condition goes unnoticed. The rot spreads from these diseased tubers to neighbouring healthy ones and the loss of the whole or a considerable part of the store frequently results.

[1] In order to save space no attempt is made in this article to give the full description of the cultural and physiological characters of the various organisms dealt with. These are readily available in the literature and where a full description of an organism is given in any paper to which reference is made an asterisk has been placed against the reference in the list of literature cited.

Symptoms of the disease. The first sign of disease is to be observed in the foliage. The leaves turn yellow or much paler green than healthy leaves and diseased plants can readily be recognised in the early stages by this character alone. At the same time they retain a rather erect position on the stem instead of spreading out horizontally and the edges exhibit a rolling towards the mid-rib. The rolling should not be confused with that of "Leaf-roll" since it is of cylindrical form rather than funnel shaped and there is entire absence of the pink or purple pigmentation characteristic of this disease. In the later stages of disease the leaves wilt and the whole plant assumes a brown to black colour and a very dejected appearance. The plants remain standing in the field in virtue of a special hardening of the three main vascular bundles of the stem. These quite early become browned for a considerable distance up the stem and are so hard that a sharp knife is needed to cut them transversely. The name "Blackleg" is derived from a characteristic blackening of the tissues of the stem commencing in the underground portion and extending to a height of two to three inches above the soil level. In the lower portion of the stem the pith is completely rotted away leaving a cavity in which fungal mycelium, easily recognised as that of saprophytic forms, is frequently present. The tissues in the upper portion of the stem appear sound except for the browning of the vascular bundles above mentioned and for a water-logged appearance of the pith for a centimetre or so above the upper limit of the cavity caused by the destruction of the pith cells of the lower portion. If the attack has occurred early in the season no tubers or only very small ones will have formed and anchorage in the soil is so slight that the least pull is sufficient to lift the plant. If, however, the plant is attacked after the tubers have well formed these will be infected by the organism through the vascular system of the underground stems. Such infected tubers will in warm moist weather rot off completely in the soil, but it more frequently happens that at the time of "lifting" the organism has not advanced from the vascular system and outwardly the tuber appears quite sound. These infected tubers give rise to the trouble in storage mentioned above and it is to them that the chief losses incurred through this disease are attributable. The organism may remain localised in the vascular ring of the tuber throughout the winter and the planting of diseased "sets" is believed to be the chief cause of dissemination of the disease.

The parasite. Several organisms have been described as giving rise to this disease but recent work by Morse(31) has shown that they are in all probability all strains of one species *B. atrosepticus*. Previously the name

B. phytophthorus was most frequently employed and Johnson(18) states that he isolated this organism from diseased potatoes in Ireland in 1906. His evidence however has not been published, and the differences between *B. phytophthorus* and another strain *B. melanogenes* isolated in the West of Ireland by Pethybridge and Murphy(34) are so slight that it is conceivable that they were overlooked by Prof. Johnson. The *B. melanogenes* strain was isolated by the author(32) from potatoes grown in Lancashire and it is possible that it is the only one present in the British Isles[1].

B. phytophthorus was not included in the strains examined and found to be identical by Morse but he had before him a culture of *B. melanogenes* and this under similar cultural treatment gave identical reactions with *B. atrosepticus*.

B. atrosepticus is a medium-sized bacillus with rounded ends motile by 3–6 peritrichous flagella and does not stain by Gram's method.

Distribution. This disease has been reported from all parts of the British Isles but the causal organism has been isolated only in the three instances mentioned above. It is a disease which seems to be definitely on the increase and unless some efficient means of control can be found may cause a great deal of trouble in the near future.

Host plants. Carruthers(9) isolated an organism from beans (*Vicia Faba*) which he believed to be identical with *B. atrosepticus* and the author has found that this organism is strongly parasitic towards *Vicia Faba*, and Appel(1) has shown that besides beans, carrots, turnips, cucumbers, tomatoes, sugar beet and mangolds are also liable to attack.

Brown Rot of Potato and Tomato.

A disease which has been very fully described by E. F. Smith(42) in which the vessels of potatoes and tomatoes become blocked with bacteria,

[1] Johnson has stated (20) that he has isolated three other organisms from diseased potato plants in Ireland, namely, *B. caulivorus*, *B. solanincola* and Kramer's bacillus to which Migula gave the name *B. solaniperda*, no evidence in support of the identification is however produced. *B. caulivorus* has been considered by Laurent (24) to be identical with *B. fluorescens liquefaciens* the pathogenicity of which still remains rather doubtful and is being investigated at the present time in this laboratory; it was found that from a prick infection with *B. atrosepticus* of a bean stem without special precautions to sterilise the epidermis the resulting rot gave a pure culture of a fluorescent bacillus and it is possible that whenever *B. fluorescens* has been isolated from diseased plants its existence is a saprophytic one following initial attack by some other organism. The pathogenicity of *B. solanincola* has been called in question by Smith (42) and there is some doubt expressed by Appel (2) as to whether the experiments of Kramer (23) are sufficient to establish the pathogenicity of his bacillus also.

an accompanying brown stain rendering the vascular bundles clearly discernible through the cortical tissue. The organisms enter the potato tuber through the stolon and produce there a brown stain in the vascular ring. From this the disease spreads as a brown rot into the storage tissue which is finally completely destroyed.

Massee (28) states that the disease has been recorded from several districts in the North of England and in Scotland. He gives a drawing which is strongly suggestive of Smith's disease and it is highly probable that the disease exists in this country, though in the absence of any evidence of the isolation of the causal organism it is still uncertain whether the English disease is etiologically identical with the American.

Iris Rot.

The leaves become yellow, wilt and die. The underground portion of the plant becomes completely rotted. Massee (29) states that the disease is quite common in this country and the author has seen it in the beds at the Chelsea Physic Garden though the causal organism has not been isolated in this country. van Hall (15) found *B. omnivorus* and two species of *Pseudomonas* responsible for the disease on the continent.

Yellow Disease of Hyacinth.

Wakker's Disease of Hyacinth due to *P. Hyacinthi* and fully described by Smith (42) occurs in our nurseries but, so far, not to any great extent. The organism was isolated by the writer from a diseased corm, recently imported from Holland, growing in one of our large nurseries in 1915 and was found to give all the characteristic reactions of *P. Hyacinthi* and to give rise to the characteristic symptoms of the disease in a corm of Roman Hyacinth (*Hyacinthus orientalis*) when the cut surface of this was smeared with a loopful of the organism from an agar slope.

Black Rot of Cabbage and other Cruciferous Plants.

The bacteria enter the leaf through the water pores causing a yellowing of the neighbouring tissue. They pass then to the veins, which become strongly blackened, and travel by way of the vascular bundles to the root which may become almost entirely hollowed out by the rot. The disease is also spread by slugs and this is probably the more usual method of infection.

The symptoms of the disease and a very full account of it are given by Smith (42). The disease was observed in England in 1901 (30) and investigated by Potter (38) who isolated a *Pseudomonas* and in Ireland in 1910 by Johnson and Adams (19) who state that they isolated *P. campestris* though their published evidence is insufficient to establish this identity. Miss Doidge (13) has found *P. campestris* in diseased cabbages raised from English seed.

The parasite. *P. campestris* is a short rod-shaped organism with rounded ends, motile by means of a simple polar flagellum.

Host plants. Practically all members of the Cruciferae are attacked by this organism, cabbages, brussels-sprouts, turnips, swedes, radish, rape, etc.

Bacterial Blight of Fruit Blossoms.

The blighting of pear and other blossoms and the consequent loss of fruit has long been recognised as one of the worst evils with which the fruit grower is beset. This had almost universally been considered to be due to the effects of frost or of cold winds until the matter was investigated by Barker and Grove (3). That these agencies, especially the former, do cause a considerable amount of loss is indisputable, but probably the greater part of the blossom blight is due to bacterial infection. The bacterial origin of the disease was discovered in 1913 by Barker and Grove who succeeded in obtaining in pure culture from diseased blossoms an organism which readily reproduced the diseased condition when placed upon or injected into various parts of flower buds borne upon healthy young shoots.

Symptoms of the disease. The first symptoms of disease vary considerably in individual flowers according to the flower part first attacked. Infection may take place in the sepals before the opening of the flower bud. These first turn grey at the tips and soon commence to blacken downwards towards the flower stalk: subsequent infection of this rapidly leads to death of the whole bud. The same result is often to be observed if the petals receive the infection, but in this case it frequently happens that the attacked petals fall prematurely before the other flower parts have become involved and the young fruit may then set normally. The receptacle frequently bears the onset of the disease, and being an internal structure the diseased condition is liable to be overlooked, the resulting failure of the fruit to set being attributed to lack of fertilisation. Small greyish-black spots appear which in a short time spread over the whole receptacle and the disease is conveyed to the ovary. Finally, the disease

may originate in the stigma, this becomes discoloured and the disease rapidly passes down the style to the ovary. Attacked blossoms showing very little obvious sign of disease may remain attached to the fruit spur for some considerable time and only fall when the tree is shaken by wind or otherwise, the slightest shaking being sufficient to bring down a shower of blossoms in all stages of development. After the fall of the flowers of a truss the spur may remain as a barren stump or may die back, this latter being in all probability due to subsequent attack by fungi such as *Nectria ditissima* or *Sclerotinia fructigena* since inoculation experiments with the causal organism on woody branches showed only slight damage. The organism may remain viable over the winter in the tissues of the spur left standing on the branch and thus may carry the disease over to the following spring.

The parasite. The causal organism is a medium sized rod with rounded ends motile by 2–5 polar flagella with lophotrichous arrangement. It stains well with the usual stains and is Gram positive. The cultural and physiological have been investigated by the above named authors and amplified by Miss E. M. Doidge(12) who obtained a culture of their organism from Prof. Barker in order to make comparison of this organism with one isolated by her from diseased pear blossoms in South Africa. Miss Doidge found certain marked differences between the two organisms which seem to justify the founding of two separate species, but, in view of the work of Morse(31), which showed that quite as marked differences may disappear when two organisms are "trained" on the same media for some considerable time, it would seem desirable that treatment on the same lines should be applied to these two strains producing Blossom Blight before arriving at a definite separation. Miss Doidge's paper does not make it clear whether any previous training had been submitted to the two organisms before the parallel series of cultures was made.

Host plants. Barker and Grove found all the varieties of pear grown at the Long Ashton Research Station were susceptible to the disease, but that some were much more so than others; Beurre d'Amanlis and Catillac being specially susceptible. The bacillus has also been isolated from certain varieties of apples, plums and cherries. Barker(4) has also isolated an organism from gooseberry bushes which is believed to be identical with the organism causing pear blossom blight though no cultural evidence of this has as yet been given.

Distribution. The organism has been isolated from affected flowers from Gloucestershire, Devon, Middlesex, Sussex, Essex, Staffordshire and

Herefordshire, and is probably wide-spread throughout the country although no statistics are available as to its occurrence in the North or in Ireland.

Bacterial Blight of Tomatoes.

Symptoms. When the tomato is quite small a minute blackish patch appears at the base of the style and from this focus the disease spreads until finally the whole fruit is reduced to a blackish soft decayed mass. It has been stated(27) that infection takes place during the flowering period and that needle prick infection from diseased tissue will set up disease in a healthy tomato.

In view of the fact that Brooks(8) holds that bacteria are not the primary cause of disease the matter badly needs further investigation in this country.

Leaf Spot Disease of Orchid.

Prof. Potter has described a bacterial blister on the leaves of *Odontoglossum uro-skinneri*(39), the epidermis being raised by an accumulation of brown mucilaginous substance caused by the action of some organism which is not fully described.

Bacterial Disease of Pisum sativum.

An interesting closed bacterial disease somewhat analogous to the closed smut of barley. The organism is present in the cotyledons of the seed, germinates with the seed, enters the plumule and passes up the stem of the plant in the motile stage and is to be found in the seed and the seed pod. It has been investigated by Miss D. M. Cayley(10) at the John Innes Horticultural Institution where for some years it has caused much trouble.

Symptoms of the disease. In the worst cases the badly infected seed fails to germinate, but usually the primary shoot develops from the infected seed, soon turns brown at the tip, aborts, and lateral shoots develop prematurely to take its place. Sometimes on the youngest shoots brown streaks are to be seen but otherwise there is no sign of disease until the flowering period. The disease develops most rapidly in hot dry weather, the stem becomes brownish and has a water-soaked appearance, brown longitudinal streaks appear on the stem at the base of the petioles, the leaves become spotted and yellowish with darkened veins and the surface of the pod is sometimes roughened or embossed. Except in bad cases the plants grow to full height, flower and set a fair crop of seed but

on examination of the seed the cotyledons are found to have brown spots or bacterial cavities at the centre of each.

The parasite. P. seminum is a large rod motile by a single polar flagellum. It stains well with the usual stains and is Gram positive. On solid media it readily forms oval bodies which may occur strung together in chains giving the appearance of beaded rods. These oval bodies are thought by Miss Cayley to be involution forms which contain certain particles, these stain as spores and are believed to be spores though their germination has not as yet been observed.

Host plants. At present the disease has been found only in *Pisum sativum*, all varieties of which would seem to be susceptible to more or less extent. The most susceptible varieties on the soil of the John Innes Institution being Ne Plus Ultra and Duke of Albany. The dwarf earlies Chelsea Gem and Little Marvel are also specially susceptible, while Sutton's Improved Petit Pois has proved on this soil to be least affected. In this variety, however, only about 50 per cent. of healthy plants were obtained.

Distribution. The disease is believed to be fairly common, at least throughout the South of England, and to be steadily on the increase. Miss Cayley reports that it is each year becoming more difficult to find seed for experimental purposes which can be relied upon to be free from this disease.

Potato Scab.

The well-known corky patches or scabs on the skin of the potato are the result of the response of the cortical cells of the tuber to the stimulus of an invading organism and not, as has been frequently stated, the result of mechanical injury or of the attack of wire-worms or other animal parasites. In its defence against the parasite the tissue of the potato is so far successful as to limit the attack to a few of the outer layers of cells only. Successive layers of cork are formed as the organism penetrates into the tissue and these may extend to a depth of half a centimetre, but the internal storage cells remain unaffected. Bolley (6) isolated a small bacterium from scabbed potatoes in Indiana and with it produced scabs by artificial infection. Later however (7) he expressed doubt as to whether this organism is responsible for the disease under natural conditions, since, in subsequent efforts, he was unable to confirm his original work, but had no difficulty in showing that the usual cause of the disease is an organism which was isolated by Thaxter (48) and described as *Oospora scabies*. The disease was included amongst those

of fungal origin until 1912 when Cunningham (11) showed the relation of *Oospora scabies* to the bacteria. In 1914 this organism was referred by Lutman and Cunningham (26) to *Actinomyces chromogenus* a species previously described by Gasperini (14).

Lutman and Cunningham have obtained four strains of *Actinomyces chromogenus* Gasperini from scabbed potatoes in America. Whether the cause of the disease is the same in this country is still uncertain, and the investigation of this matter is one of the many problems in plant bacteriology which call for elucidation. It is very unsatisfactory that a disease so long-established in the country should not yet have been attributed to a definite organism.

Crown Gall.

An intumescence disease on the roots of various plants has been fully described by Smith and Townsend (44). The same symptoms of disease have been found on the roots of Paris daisy (25) (*Chrysanthemum frutescens* L.) grown at Kew; although the organism has not been isolated here there seems little reason to doubt that the disease is etiologically identical with that in America.

A very large number of host plants have been found to be affected with this disease. In this country they have been observed also in plum, rose, raspberry and loganberry and in the United States on peach, apricot, almond, prune, apple, cherry, poplar, chestnut and blackberry.

So-called Physiological Diseases.

A number of obscure diseases have been attributed to a derangement of the normal physiological condition of the plant without the intervention of a parasite. This is in all probability a lame way of hiding our ignorance, and the possibility of the existence of ultra-microscopic organisms in these cases should always be kept in the fore-front.

The "Leaf-roll" disease of potatoes has been investigated thoroughly by Quanjer (40) who finds a pathological condition of the phloëm elements of the stem to which he ascribes the visible symptoms of disease. The facts that the disease is communicable to a scion by grafting a disease-bearing stock and *vice versa* and that it is transmissible through the soil would seem to point strongly to a bacterial origin of the disease, and further investigation is advisable before any cause other than a parasitic one is accepted for this disease.

The "Mosaic Disease of Tomato" has also been shown to be transmissible through the soil and the same argument applies in this case also.

"Sprain" in potatoes is another of these obscure phenomena and there would seem to be several different kinds of "sprain." One form has been attributed (25), upon what evidence the author is unable to discover, to an arrested condition of Winter-Rot. Another form which has been described as "Internal Disease" by Horne (17) is believed to be similar to a disease which the writer is now investigating and which he has found to be due to a bacterial parasite (33).

"Silver-Leaf Disease" in plum has also been suggested as due to a bacterial parasite (5) and Smolâk (47) observed bacteria in the vessels of the leaf although the relationship of these to the disease was not determined.

The need for further investigation of bacterial diseases and for more workers in the field is vital, how vital has recently been shown by the discovery in America of two diseases of cereal crops, a bacterial blight of Barley (21) and a disease of Wheat (46); the latter is stated to be "not as destructive as the rusts but more destructive than the smuts and very likely more difficult to control." The means of control in bacterial disease are very difficult and necessitate a vast amount of research. Possible lines of attacking the problem have been so ably set out by Smith (45) in his *Conspectus of Bacterial Diseases* that little can be added in the present state of our knowledge.

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"INTERNAL RUST SPOT" DISEASE OF THE POTATO TUBER.

(*Preliminary communication.*)

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Through the courtesy of Mr A. D. Cotton of the Kew Gardens Pathological Laboratory the writer has obtained from two farms in the South of England specimens of potatoes suffering from a disease which had been diagnosed as of bacterial origin. From one farm at Dunstable the disease was so bad as to render the crop quite unsaleable, over 90 per cent. of the tubers being badly affected, and from the other farm at Christchurch came the report that at least 33 per cent. of the tubers had to be wholly rejected, so that it is safe to assume that upwards of 50 per cent. were affected to some extent.

The symptoms of the disease were thought at first to indicate *Pseudomonas solanacearum* as the cause. On further examination of diseased tubers, however, appearances were discovered which were quite different from those described by Smith(6) as characteristic of the attack of this organism. There is, for instance, no appearance whatever of a milky slime in the affected part of the tissue and the potatoes do not rot off during the winter. The tuber remains perfectly hard and appears sound except for a few brown patches on the skin and a certain appearance of "scabbiness." Around some of the lenticels the skin is raised and cracked, and on removal of this there is revealed a darkened sunken area of corky tissue resembling somewhat the appearance of an old *Spongospora* scab.

On cutting a tuber discoloured patches of tissue of varying size are found scattered over the surface. These are typically of a brownish-red colour resembling that of iron rust or, perhaps, more nearly that of ginger-bread. They are distributed promiscuously through the tuber and only occasionally seem to have developed in proximity to the vascular

ring. In some of the first potatoes examined the patches had a greenish-black water-soaked appearance and seemed to have arisen from the vascular bundles suggesting *P. solanacearum* as the causal organism; it is, however, probable that the water-logging is secondary being due to the presence in the diseased tissue of certain saprophytic bacteria.

The size of the patches varies enormously from a small island of brown tissue of the size of a pin's head to an irregular-shaped patch spreading through several centimetres of the tissue. In certain cases the large patches can be traced to lenticels which show the "scabbed" appearance described above, indicating that the exciting cause has found entry into the tuber through the lenticel and that "the scabbing" is the result of its action on the surrounding cells.

Attempts to prepare diseased tissue for microtome sectioning have, so far, been unsuccessful, strong lignification of the walls of the attacked cells making them very difficult of penetration by fixing and other fluids. Hand sections through a small patch show a group of normal cells packed with starch grains and surrounded by a ring of brown tissue. In the brown zone the cells have thickened walls which stain red with phloroglucin and this coupled with their resistance to cutting has led to the conclusion that they are strongly lignified. As contents, these cells show brown protoplasm in which starch grains may be embedded or the starch may be replaced by globules of various shape and size which appear to be of oil or possibly of tannin. In certain of the cells there may be recognised masses of small bacteria intermingled with the starch grains. In some sections, however, bacteria could not be distinguished, either by motility or shape, from minute globules of the fat-like substance. The brown ring which gives the characteristic appearance to the patch is surrounded by a zone of colourless cells in which the starch has entirely disappeared and given place to more or less colourless oily globules. Frequently a small patch is found to be cut off from this zone of starch-free cells by the formation of a typical cork callus.

Sections through one of the larger patches show essentially the same cell characters as described above except that the inner cells are much disorganised, or they may have dried up and broken down leaving a cavity surrounded by cells with the characteristic brown and lignified walls.

By inoculations of diseased tissue into sterile potato broth an organism has with difficulty been obtained and shown to be the cause of the disease. Inoculations of sterile potato slices using pure cultures of the organism have repeatedly given successful infections, and from the diseased area,

which was thoroughly characteristic of the disease in the original tuber, the organism has again been isolated. Occasionally inoculations of potato broth with pieces of the original diseased tissue have failed entirely to produce turbidity in the broth. This sterility is believed to be due to the weak parasitism of the causal organism, the plant tissue having by cork formation confined the parasite within narrow limits in which it has died out during the winter.

The symptoms of the disease here dealt with correspond exactly with those described by Horne (3) for "Internal Disease" and by Mayer (4), Frank, A. B. (1), Sorauer and Rörig (5) and Frank, B. (2) under the names "Buntwerden," "Eisenfleckigkeit," "Buntheit," or "Stockfleckigkeit." There seems little doubt that these workers have described one and the same disease. All the German workers have come to the conclusion that the disease is not of parasitic origin and have sought for the cause in the physiological condition of the soil. Horne alone suggests that further search for a parasite might be advisable. The question of the identity of their disease with the one dealt with in this paper, for which the name "Internal Rust Spot" is suggested, is therefore of some considerable interest. Further discussion of this relationship and a full description of the causal organism of the "Internal Rust Spot" disease are reserved for a later paper.

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